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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
39TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE
Controversies in Home Respiratory Care
Part II

- Airway Clearance: New Techniques, Indications, and Recommendations
- ICU to Home: Patient Selection and Discharge Planning
- Physical Reconditioning of Patients with Respiratory Diseases
- Noninvasive Positive-Pressure Ventilation at Home
- Indications and Criteria for Decannulation and Transition from Invasive to Noninvasive Long-Term Ventilatory Support
- Use of Negative Pressure Ventilation, Rocking Beds, and Pneumobelts
- Home Mechanical Ventilatory Assistance for Infants
- Collaborative Self-Management Strategies for Patients with Respiratory Disease

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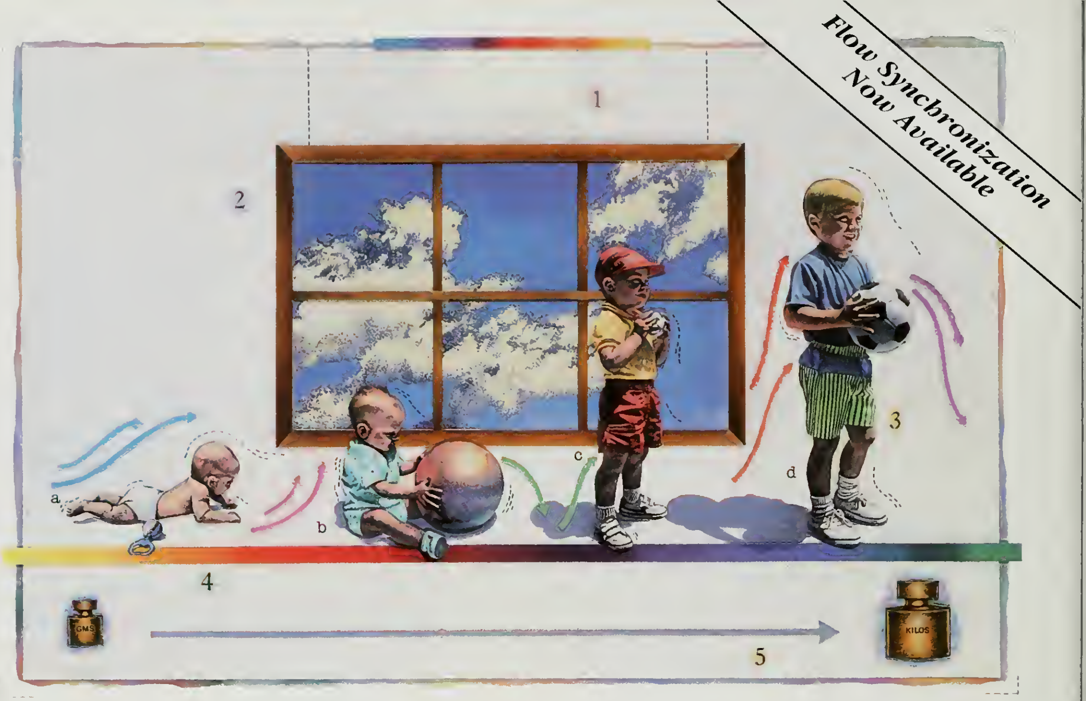

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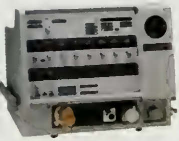

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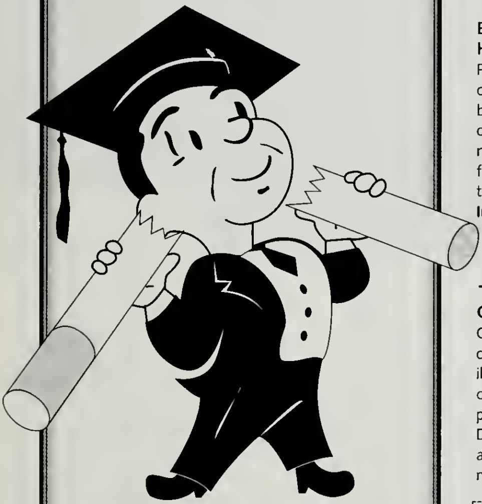

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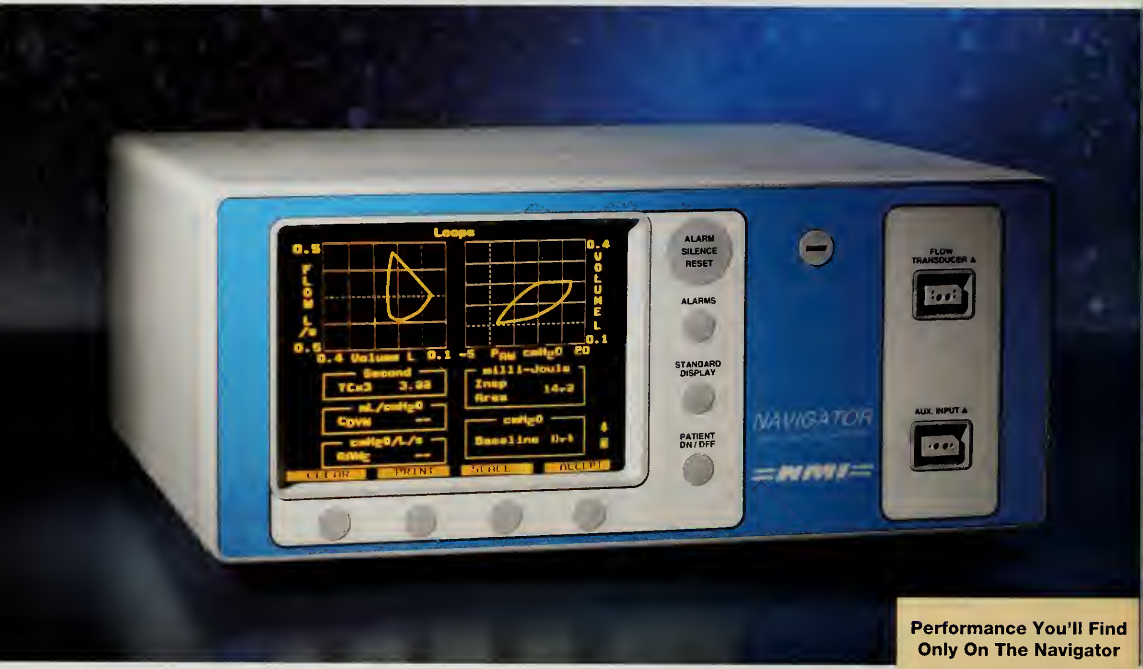

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Psychosocial Factors and Prognosis in Established Coronary Artery Disease: The Need for Research on Interventions (editorial)—RB Williams, MA Chesney. JAMA 1993;270(15):1860. (Pertains to Frasure-Smith et al paper abstracted on Page 421.)

The Case for Standardized Terminology: Oxygen "Saturation" Values Can Trick the Unwary and Lead to Clinical Misjudgment (letter)—DK Cope, AE Taylor. Crit Care Med 1993;21(5):805.

Accurate Assessment of Right Ventricular Function in Acute Respiratory Failure—C Her, DE Lees. Crit Care Med 1993;21(11):1665.

OBJECTIVE: Since right ventricular ejection fraction is highly dependent on afterload, right ventricular ejection fraction may not reflect right ventricular contractile function in acute respiratory failure. Despite a severe reduction in right ventricular ejection fraction, the right ventricle may be able to generate pressure output that is sufficient enough to maintain an adequate distribution of pulmonary perfusion. We tested this hypothesis by assessing the correlation between the right ventricular ejection fraction and the right ventricular end-systolic pressure-volume relationship, and by assessing the correlations between right ventricular ejection fraction and the physiologic dead space/tidal volume ratio and between the physiologic dead space/ tidal volume ratio and the right ventricular end-systolic pressure-volume relationship. DESIGN: Prospective study. SETTING: University hospital intensive care unit (ICU). PATIENTS: Twenty-one patients with acute respiratory failure. MEASUREMENTS & MAIN RESULTS: The physiologic dead space/tidal volume ratio was used as an index of the distribution of pulmonary perfusion. Right ventricular ejection fraction was measured by the thermodilution method. Right ventricular end-diastolic volume index was obtained from the stroke volume index divided by the right ventricular ejection fraction. Right ventricular end-systolic volume index was calculated as the difference between the right ventricular end-diastolic volume index and the stroke volume index. Pulmonary arterial dicrotic notch pressure was used as an estimate of right ventricular end-systolic pressure. Data were collected at baseline and after one or two alterations in preload to define the right ventricular end-systolic pressure-volume relationship. There was no correlation between the right ventricular ejection fraction and the slope of the right ventricular end-systolic pressure-volume relationship line. No correlation was found between the right ventricular ejection fraction and the physiologic dead space/tidal volume ratio. There was a hyperbolic curvilinear relationship between the physiologic dead space/ tidal volume ratio and the slope of the right ventricular end-systolic pressure-volume relationship line ($r^2 = 0.82$, $p < 0.0001$). When the patients were divided into two groups based on the slope of the right ventricular end-systolic pressure-volume relationship line, the physiologic dead space/tidal volume ratio was lower in the group with a high slope of the right ventricular end-systolic pressure-volume relationship line ($p < 0.0001$). There was no difference in other hemodynamic data between the two groups. CONCLUSIONS: These data suggest that in acute respiratory failure, the right ventricular ejection fraction does not reflect right ventricular performance.

Survival in the Elderly after Out-of-Hospital Cardiac Arrest—MJ Bonnin, PE Pepe, PS Clark Jr. Crit Care Med 1993;21(11):1645.

OBJECTIVES: To evaluate the survival prognosis for the elderly (≥ 70 yrs of age) after out-of-hospital cardiac arrest in a large urban center, and to identify any specific differences in survival factors relative to those adults < 70 yrs of age. DESIGN: The study was a prospective, inception cohort study. SETTING: An urban population of approximately 2,000,000, served by one centralized municipal emergency medical services system. PATIENTS: All 986 adult victims (367 elderly and 619 younger patients) of primary cardiac arrest attended by the emergency medical services system over a 12-month period. INTERVENTIONS: Not applicable. MEASUREMENTS & MAIN RESULTS: All victims of out-of-hospital cardiac arrest occurring within a single, large, urban municipality were studied over a 12-month period. Each event was analyzed for age, sex, witnesses, bystander cardiopulmonary resuscitation, presenting electrocardiographic rhythm, paramedic response time, scene time, return of spontaneous circulation (pulses), and electrocardiographic rhythm on hospital arrival. Outcomes evaluated included inhospital admission (resuscitation) and successful discharge from the hospital (survival). Patients were followed until death or discharge from the hospital. Of 367 elderly cardiac arrest victims, 81 (22%) patients were successfully resuscitated and 24 (7%) patients survived. However, of 119 (32% of all elderly patients) patients who presented with ventricular fibrillation/tachycardia, 48 (40%) patients were resuscitated and 17 (14%) patients survived. These 17 patients with ventricular fibrillation/tachycardia accounted for 71% of all elderly survivors. During the same study period, there were 619 adult primary cardiac arrest victims < 70 yrs of age, 160 (26%) of whom were resuscitated and 73 (12%) of whom survived. Among the younger patients, 296 (48%) patients presented with ventricular fibrillation/tachycardia, of whom 110 (37%) were resuscitated and of whom 60 patients (20%) survived. Within the context of this study, survival rates for younger and older ventricular fibrillation/tachycardia patients were not significantly different. Also, among survivors,

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there were no other major differences in terms of established survival determinants. CONCLUSIONS: Survival chances for the elderly after out-of-hospital cardiac arrest are not bleak, and are reasonable if ventricular fibrillation/tachycardia is the presenting rhythm. Survival determinants are similar for younger and older adults.

Increased Pulmonary Perfusion Worsens Ventilation-Perfusion Matching—KB Domino, BL Eisenstein, T Tran, MP Hlastala. Anesthesiology 1993;79(4):817.

BACKGROUND: Severe exercise and administration of vasopressors may adversely affect pulmonary gas exchange in humans. The role of increases in pulmonary perfusion in worsening ventilation-perfusion ($\dot{V}_A/\dot{Q}$) relationships is unclear, however, because concomitant changes in ventilation and alveolar gas composition occur. The purpose of this study was to determine whether increasing of lobar blood flow increased $\dot{V}_A/\dot{Q}$ heterogeneity in the absence of changes in respiratory parameters. METHODS: Six pentobarbital-anesthetized dogs underwent bilateral thoracotomies, left upper lobectomy, and placement of an electromagnetic flow probe on the left lower lobe (LLL) pulmonary artery, and catheters were inserted into the LLL pulmonary artery distal to the flow probe and confluent trunk of the LLL pulmonary vein. A bronchial divider was inserted to allow separate ventilation of the right lung and LLL. Blood flow to the LLL ($\dot{Q}_{LLL}$) was increased in random order to two and three times baseline blood flow by opening an arteriovenous fistula and partially occluding the right pulmonary artery. Minute ventilation and alveolar P_{CO_2} of the lobe were unchanged due to use of constant tidal volume and respiratory rate and inspiration of variable amounts of carbon dioxide. $\dot{V}_A/\dot{Q}$ distributions of the LLL were obtained using the multiple inert gas elimination technique. The tracer inert gas arterial-alveolar difference ([a–A]D) area was used to assess $\dot{V}_A/\dot{Q}$ mismatch. RESULTS: Increasing $\dot{Q}_{LLL}$ increased mean pulmonary artery pressure in the LLL (LLL P_{pa}). The P_{O_2} of the LLL pulmonary venous blood remained unchanged, as the mixed venous oxygen tension ($P_{\bar{v}O_2}$) was markedly increased. $\dot{V}A/\dot{Q}$ inequality was increased, indicated by a 40% increase in the [a–A]D area when $\dot{Q}_{LLL}$ was increased to two times greater than baseline $\dot{Q}_{LLL}$ and a 58% increase in the [a–A]D area with three times greater than baseline $\dot{Q}_{LLL}$. The [a-A]D area was highly correlated with the lobar blood flow ($r = 0.97$) and LLL P_{pa} ($r = 0.97$). CONCLUSIONS: Marked increases in lobar blood flow and P_{pa} worsened pulmonary gas exchange. The degree of impairment was correlated with the degree of increase in lobar perfusion. However, increased lobar perfusion did not affect LLL pulmonary venous blood oxygenation because the decrease in P_{O_2}, due to increased $\dot{V}_A/\dot{Q}$ mismatch, was opposed by an increase in P_{O_2}, due to increased $P_{\bar{v}O_2}$.

Regular Inhaled Salbutamol and Airway Responsiveness to Allergen—DW Cockcroft, CP McParland, SA Britto, VA Swystun, BC Rutherford. Lancet 1993;342:833.

Regular inhaled β_2 agonist causes tolerance to the acute protective effect of β_2, agonist against bronchoconstriction induced by chemical stimuli such as AMP, histamine, and methacholine. We examined a more clinically relevant stimulus, inhaled allergen, in a double-blind, cross-over, random-order trial in 13 mild atopic asthmatics, who had not used β_2 agonist for at least 4 weeks. We compared regular inhaled salbutamol (200 μg four times daily for 2 weeks) with placebo (2 weeks) for effects on bronchodilator response, baseline methacholine, and allergen airway responsiveness, and on the acute protective effect of salbutamol against both stimuli. Baseline forced expiratory volume in 1 s (FEV_1), bronchodilator response, and methacholine responsiveness were the same during both treatment periods. After regular salbutamol, the allergen PC_{20} (provocation concentration producing a 20% FEV_1 decrease) fell by 0.91 (SD 0.66) ($p = 0.0009$) doubling doses, and the protective effects of salbutamol on methacholine and allergen were both significantly reduced ($p = 0.026$ and 0.025, respectively). Taking into account the reduced baseline allergen PC_{20}, the post-salbutamol allergen PC_{20} was almost 2 doubling doses (1.94 [1.43], $p < 0.01$) lower during salbutamol treatment. Thus, 2 weeks of regular inhaled salbutamol increased airway responsiveness to allergen but not to methacholine, and caused tolerance to the protective effect of salbutamol on bronchoconstriction induced by both stimuli. These effects of inhaled β_2 agonist provide further evidence to support detrimental effects of their regular use.

Guidelines and Levels of Care for Pediatric Intensive Care Units—American Academy of Pediatrics: Committee on Hospital Care. Crit Care Med 1993;21:1077.

OBJECTIVE: To provide recommendations that are current and attainable for the organization, personnel, equipment, facilities, and services necessary for pediatric intensive care units (ICUs). DATA SOURCES: Review of the medical literature and consensus of practitioners in the field, including physicians from multiple specialties, critical care nurses, and respiratory therapists. SETTING: Annual meetings of the Society of Critical Care Medicine and American Academy of Pediatrics. PARTICI-

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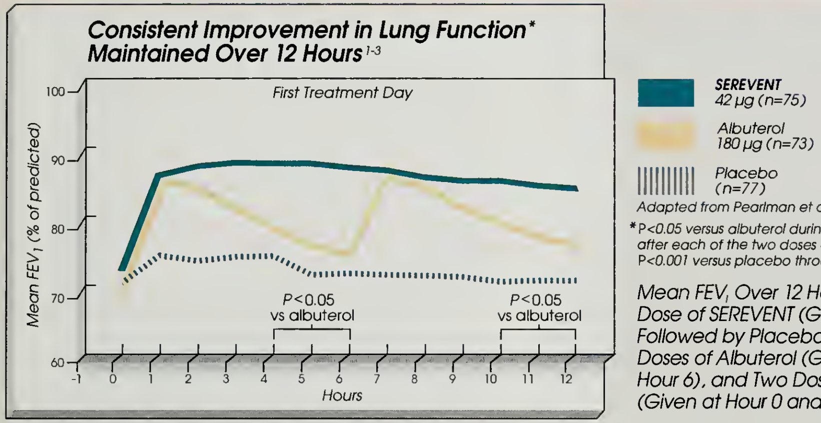
SEREVENT: The Next Dimension in Inhaled Bronchodilator Therapy


Consistent Improvement in Lung Function* Maintained Over 12 Hours[1-3]

Adapted from Pearlman et al.[1]

*$P<0.05$ versus albuterol during the periods 4 to 6 hours after each of the two doses of albuterol. $P<0.001$ versus placebo throughout the 12-hour period.

Mean FEV$_1$ Over 12 Hours After the First Dose of SEREVENT (Given at Hour 0 Followed by Placebo at Hour 6), Two Doses of Albuterol (Given at Hour 0 and Hour 6), and Two Doses of Placebo (Given at Hour 0 and Hour 6)

- Proven for 12 weeks,[1,2] maintained over 12 months.[4]
- SEREVENT is indicated for prevention and maintenance treatment of asthma (including nocturnal asthma) in patients 12 years of age and older who require regular treatment with inhaled, short-acting β_2-agonists.
- Convenient twice-daily[†] dosing.
- Outstanding side effect profile comparable to albuterol.
  - The most common drug-related adverse events reported in clinical trials were headache (10%), tremor (3%), and cough (3%).[2]

New, Twice-Daily

Serevent®

(salmeterol xinafoate)

Inhalation Aerosol

Morning and Evening Inhalation for Active Days and Restful Nights

[†] Dosing should be two puffs (42 µg) of SEREVENT Inhalation Aerosol, twice daily, morning and evening, approximately 12 hours apart, for the maintenance treatment of asthma. SEREVENT should not be used for the treatment of acute exacerbations.

Please consult Brief Summary of Prescribing Information on the following pages.

SEREVENT *60-Dose Institutional Package*	*NDC 0173-0467-00*
SEREVENT *120-Dose Trade Package*	*NDC 0173-0464-00*

Serevent®
(salmeterol xinafoate)
Inhalation Aerosol

BRIEF SUMMARY

Bronchodilator Aerosol
For Oral Inhalation Only

The following is a brief summary only. Before prescribing, see complete prescribing information in Serevent® Inhalation Aerosol product labeling.

CONTRAINDICATIONS: Serevent® Inhalation Aerosol is contraindicated in patients with a history of hypersensitivity to any of the components.

WARNINGS: 1. Not for Use to Treat Acute Symptoms: Watch for Increased Need for Short-Acting Beta$_2$-Agonists: Serevent® Inhalation Aerosol should not be used to relieve acute asthma symptoms. If the patient's short-acting, inhaled beta$_2$-agonist becomes less effective, e.g., the patient needs more inhalations than usual, medical evaluation must be obtained immediately and increasing use of Serevent Inhalation Aerosol in this situation is inappropriate. Serevent Inhalation Aerosol should not be used more frequently than twice daily (morning and evening) at the recommended dose. When prescribing Serevent Inhalation Aerosol, patients must be provided with a short-acting, inhaled beta$_2$-agonist (e.g., albuterol) for treatment of symptoms that occur despite regular twice-daily (morning and evening) use of Serevent.

Asthma may deteriorate acutely over a period of hours or chronically over several days. In this setting, increased use of inhaled, short-acting beta$_2$-agonists is a marker of destabilization of asthma and requires re-evaluation of the patient and consideration of alternative treatment regimens, especially inhaled or systemic corticosteroids. If the patient uses four or more inhalations per day of a short-acting beta$_2$-agonist on a regular basis, or if more than one canister (200 inhalations per canister) is used in an 8-week period, then the patient should see the physician for re-evaluation of treatment.

2. Use With Short-Acting Beta$_2$-Agonists: When patients begin treatment with Serevent Inhalation Aerosol, those who have been taking short-acting, inhaled beta$_2$-agonists on a regular daily basis should be advised to discontinue their regular daily-dosing regimen and should be clearly instructed to use short-acting, inhaled beta$_2$-agonists only for symptomatic relief if they develop asthma symptoms while taking Serevent Inhalation Aerosol (see PRECAUTIONS: Drug Interactions).

3. Serevent Inhalation Aerosol is Not a Substitute for Oral or Inhaled Corticosteroids: Patients must be warned not to stop or reduce corticosteroid therapy without medical advice, even if they feel better when they are being treated with Serevent.

4. Do Not Exceed Recommended Dose: As with other beta-adrenergic aerosols, Serevent Inhalation Aerosol should not be used in excess. Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs. Large doses of inhaled or oral salmeterol (12 to 20 times the recommended dose) have been associated with clinically significant prolongation of the QT_C interval, which has the potential for producing ventricular arrhythmias.

5. Paradoxical Bronchospasm. As with other inhaled asthma medications, paradoxical bronchospasm (which can be life-threatening) has been reported following the use of Serevent Inhalation Aerosol. If it occurs, treatment with Serevent Inhalation Aerosol should be discontinued immediately and alternative therapy instituted.

6. Immediate Hypersensitivity Reactions: Immediate hypersensitivity reactions may occur after administration of Serevent Inhalation Aerosol, as demonstrated by rare cases of urticaria, rash, and bronchospasm.

PRECAUTIONS:

General: 1. Use with Spacer or Other Devices: The safety and effectiveness of Serevent® Inhalation Aerosol when used with a spacer or other devices have not been adequately studied.

2 Cardiovascular and Other Effects: No effect on the cardiovascular system is usually seen after the administration of inhaled salmeterol in recommended doses, but the cardiovascular and central nervous system effects seen with all sympathomimetic drugs (e.g., increased blood pressure, heart rate, excitement) can occur after use of Serevent Inhalation Aerosol and may require discontinuation of the drug. Salmeterol, like all sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension; in patients with convulsive disorders or thyrotoxicosis; and in patients who are unusually responsive to sympathomimetic amines.

As has been described with other beta-adrenergic agonist bronchodilators, clinically significant changes in systolic and/or diastolic blood pressure, pulse rate, and electrocardiograms have been seen infrequently in individual patients in controlled clinical studies with salmeterol.

3. Metabolic Effects: Doses of the related beta$_2$-adrenoceptor agonist albuterol, when administered intravenously, have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis. No effects on glucose have been seen with Serevent Inhalation Aerosol at recommended doses. Administration of beta$_2$-adrenoceptor agonists may cause a decrease in serum potassium, possibly through intracellular shunting, which has the potential to increase the likelihood of arrhythmias. The decrease is usually transient, not requiring supplementation.

Clinically significant changes in blood glucose and/or serum potassium were seen rarely during clinical studies with long-term administration of Serevent Inhalation Aerosol at recommended doses.

Information for Patients: See illustrated Patient's Instructions for Use. **SHAKE WELL BEFORE USING.** Patients should be given the following information:

1. Not for Use to Treat Acute Symptoms: Serevent Inhalation Aerosol is not meant to relieve acute asthmatic symptoms. Acute symptoms should be treated with an inhaled, short-acting bronchodilator that has been prescribed by a physician for symptom relief.

2. Do Not Exceed Recommended Dose: The bronchodilator action of salmeterol usually lasts for at least 12 hours. Therefore it should not be used more often than every 12 hours.

3. Use With Other Medications: While using Serevent Inhalation Aerosol, other inhaled medicines should be taken only as directed by the physician.

4 Use with Short-Acting, Inhaled Beta-Agonists: While using Serevent Inhalation Aerosol, medical attention should be sought immediately if the short-acting bronchodilator treatment becomes less effective for symptom relief, if more inhalations than usual are needed, or if more than the maximum number of inhalations of short-acting bronchodilator treatment prescribed for a 24-hour period are needed. If the patient uses four or more inhalations per day of a short-acting beta$_2$-agonist on a regular basis, or if more than one canister (200 inhalations per canister) is used in an 8-week period, then the patient should see the physician for re-evaluation of treatment.

Patients should be cautioned regarding potential adverse cardiovascular effects, such as palpitations or chest pain, related to the use of additional beta$_2$-agonist.

5. Use of Systemic or Inhaled Steroids: Serevent® (salmeterol xinafoate) Inhalation Aerosol does not replace oral or inhaled corticosteroids; the dosage of these medicines should not be changed and they should not be stopped without consulting the physician, even if the patient feels better.

6. Use for Exercise-Induced Bronchospasm: When using Serevent Inhalation Aerosol to prevent exercise-induced bronchospasm, the dose should be administered at least 30 to 60 minutes before exercise.

Drug Interactions: ***Short-Acting Beta-Agonists:*** In the two 3-month, repetitive-dose clinical trials (n=184), the mean daily need for additional beta$_2$-agonist use was 1 to 1½ inhalations per day, but some patients used more. Eight percent of patients used at least eight inhalations per day at least on one occasion. Six percent used 9 to 12 inhalations at least once. There were 15 patients (8%) who averaged over four inhalations per day. Four of these used an average of 8 to 11 inhalations per day. In these 15 patients there was no observed increase in frequency of cardiovascular adverse events. The safety of concomitant use of more than eight inhalations per day of short-acting beta$_2$-agonists with Serevent Inhalation Aerosol has not been established. In 15 patients who experienced worsening of asthma while receiving Serevent Inhalation Aerosol, nebulized albuterol (one dose in most) led to improvement in FEV_1 and no increase in occurrence of cardiovascular adverse events.

Monoamine Oxidase Inhibitors and Tricyclic Antidepressants: Salmeterol should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants because the action of salmeterol on the vascular system may be potentiated by these agents.

Corticosteroids and Cromoglycate: In clinical trials, inhaled corticosteroids and/or inhaled cromolyn sodium did not alter the safety profile of Serevent Inhalation Aerosol when administered concurrently.

Methylxanthines: The concurrent use of intravenously or orally administered methylxanthines (e.g., aminophylline, theophylline) by patients receiving Serevent Inhalation Aerosol has not been completely evaluated. In one clinical trial, 87 patients receiving Serevent Inhalation Aerosol 42 mcg twice daily concurrently with a theophylline product had adverse event rates similar to those in 71 patients receiving Serevent Inhalation Aerosol without theophylline. Resting heart rates were slightly higher in the patients on theophylline but were little affected by Serevent Inhalation Aerosol therapy.

Carcinogenesis, Mutagenesis, Impairment of Fertility: In an 18-month oral carcinogenicity study in CD-mice, salmeterol xinafoate caused a dose-related increase in the incidence of smooth muscle hyperplasia, cystic glandular hyperplasia, and leiomyomas of the uterus and a dose-related increase in the incidence of cysts in the ovaries. A higher incidence of leiomyosarcomas was not statistically significant; tumor findings were observed at oral doses of 1.4 and 10 mg/kg, which gave 9 and 63 times, respectively, the human exposure based on rodent:human AUC comparisons.

Salmeterol caused a dose-related increase in the incidence of mesovarian leiomyomas and ovarian cysts in Sprague Dawley rats in a 24-month inhalation/oral carcinogenicity study. Tumors were observed in rats receiving doses of 0.68 and 2.58 mg/kg per day (about 55 and 215 times the recommended clinical dose [mg/m^2]). These findings in rodents are similar to those reported previously for other beta-adrenergic agonist drugs. The relevance of these findings to human use is unknown.

No significant effects occurred in mice at 0.2 mg/kg (1.3 times the recommended clinical dose based on comparisons of the AUCs) and in rats at 0.21 mg/kg (15 times the recommended clinical dose on a mg/m^2 basis).

Salmeterol xinafoate produced no detectable or reproducible increases in microbial and mammalian gene mutation *in vitro*. No blastogenic activity occurred *in vitro* in human lymphocytes or *in vivo* in a rat micronucleus test. No effects on fertility were identified in male and female rats treated orally with salmeterol xinafoate at doses up to 2 mg/kg orally (about 160 times the recommended clinical dose on a mg/m^2 basis).

Pregnancy: ***Teratogenic Effects: Pregnancy Category C:*** No significant effects of maternal exposure to oral salmeterol xinafoate occurred in the rat at doses up to the equivalent of about 160 times the recommended clinical dose on a mg/m_2 basis. Dutch rabbit fetuses exposed to salmeterol xinafoate *in utero* exhibited effects characteristically resulting from beta-adrenoceptor stimulation; these included precocious eyelid openings, cleft palate, sternebral fusion, limb and paw flexures, and delayed ossification of the frontal cranial bones. No significant effects occurred at 0.6 mg/kg given orally (12 times the recommended clinical dose based on comparison of the AUCs).

New Zealand White rabbits were less sensitive since only delayed ossification of the frontal bones was seen at 10 mg/kg given orally (approximately 1,600 times the recommended clinical dose on a mg/m^2 basis). Extensive use of other beta-agonists has provided no evidence that these class effects in animals are relevant to use in humans. There are no adequate and well-controlled studies with Serevent Inhalation Aerosol in pregnant women. Serevent Inhalation Aerosol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Use in Labor and Delivery: There are no well-controlled human studies that have investigated effects of salmeterol on preterm labor or labor at term. Because of the potential for beta-agonist interference with uterine contractility, use of Serevent Inhalation Aerosol during labor should be restricted to those patients in whom the benefits clearly outweigh the risks.

Nursing Mothers: Plasma levels of salmeterol after inhaled therapeutic doses are very low (85 to 200 pg/mL) in humans. In lactating rats dosed with radiolabeled salmeterol, levels of radioactivity were similar in plasma and milk. In rats, concentrations of salmeterol in plasma and milk were similar. The xinafoate moiety is also transferred to milk in rats at concentrations of about half the corresponding level in plasma. However, since there is no experience with use of Serevent Inhalation Aerosol by nursing mothers, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when salmeterol xinafoate is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of Serevent Inhalation Aerosol in children younger than 12 years of age have not been established.

Geriatric Use: Of the total number of patients who received Serevent Inhalation Aerosol in all clinical studies, 241 were 65 years and older. Geriatric patients (65 years and older) with reversible obstructive airway disease were evaluated in four well-controlled studies of 3 weeks' to 3 months' duration. Two placebo-controlled, crossover studies evaluated twice-daily dosing with salmeterol for 21 to 28 days in 45 patients. An additional 75 geriatric patients were treated with salmeterol for 3 months in two large parallel-group, multicenter studies. These 120 patients experienced increases in AM and PM peak expiratory flow rate and decreases in diurnal variation in peak expiratory flow rate similar to responses seen in the total populations of the two latter studies. The adverse event type and frequency in geriatric patients were not different from those of the total populations studied.

No apparent differences in the efficacy and safety of Serevent Inhalation Aerosol were observed when geriatric patients were compared with younger patients in clinical trials. As with other beta$_2$-agonists, however, special caution should be observed when using Serevent Inhalation Aerosol in elderly patients who have concomitant cardiovascular disease that could be adversely affected by this class of drug. Based on available data, no adjustment of salmeterol dosage in geriatric patients is warranted.

Serevent® (salmeterol xinafoate) Inhalation Aerosol

ADVERSE REACTIONS: Adverse reactions to salmeterol are similar in nature to reactions to other selective beta$_2$-adrenoceptor agonists, i.e., tachycardia; palpitations; immediate hypersensitivity reactions, including urticaria, rash, bronchospasm (see WARNINGS); headache; tremor; nervousness; and paradoxical bronchospasm (see WARNINGS).

Two multicenter, 12-week, controlled studies have evaluated twice-daily doses of Serevent® (salmeterol xinafoate) Inhalation Aerosol in patients 12 years of age and older with asthma. The following table reports the incidence of adverse events in these two studies.

Adverse Experience Incidence in Two Large 12-Week Clinical Trials*

Adverse Event Type	Percent of Patients		
	Placebo n=187	Serevent 42 mcg b.i.d. n=184	Albuterol 180 mcg q.i.d. n=185
Ear, nose, and throat			
Upper respiratory tract infection	13	14	16*
Nasopharyngitis	12	14	11
Disease of nasal cavity/sinus	4	6	1
Sinus headache	2	4	<1
Gastrointestinal			
Stomachache	0	4	0
Neurological			
Headache	23	28	27
Tremor	2	4	3
Respiratory			
Cough	6	7	3
Lower respiratory infection	2	4	2

* The only adverse experience classified as serious was one case of upper respiratory tract infection in a patient treated with albuterol

The table above includes all events (whether considered drug related or nondrug related by the investigator) that occurred at a rate of over 3% in the Serevent Inhalation Aerosol treatment group and were more common in the Serevent Inhalation Aerosol group than in the placebo group.

Pharyngitis, allergic rhinitis, dizziness/giddiness, and influenza occurred at 3% or more but were equally common on placebo. Other events occurring in the Serevent Inhalation Aerosol treatment group at a frequency of 1% to 3% were as follows:

Cardiovascular: Tachycardia, palpitations.
Ear, Nose, and Throat: Rhinitis, laryngitis.
Gastrointestinal: Nausea, viral gastroenteritis, nausea and vomiting, diarrhea, abdominal pain.
Hypersensitivity: Urticaria.
Mouth and Teeth: Dental pain.
Musculoskeletal: Pain in joint, back pain, muscle cramp/contraction, myalgia/myositis, muscular soreness.
Neurological: Nervousness, malaise/fatigue.
Respiratory: Tracheitis/bronchitis.
Skin: Rash/skin eruption.
Urogenital: Dysmenorrhea.

In small dose-response studies, tremor, nervousness, and palpitations appeared to be dose related.

OVERDOSAGE: Overdosage with salmeterol may be expected to result in exaggeration of the pharmacologic adverse effects associated with beta-adrenoceptor agonists, including tachycardia and/or arrhythmia, tremor, headache, and muscle cramps. Overdosage with salmeterol can lead to clinically significant prolongation of the QT_C interval, which can produce ventricular arrhythmias. Other signs of overdosage may include hypokalemia and hyperglycemia.

In these cases, therapy with Serevent® Inhalation Aerosol and all beta-adrenergic–stimulant drugs should be stopped, supportive therapy provided, and judicious use of a beta-adrenergic blocking agent should be considered, bearing in mind the possibility that such agents can produce bronchospasm. Cardiac monitoring is recommended in cases of overdosage.

As with all sympathomimetic pressurized aerosol medications, cardiac arrest and even death may be associated with abuse of Serevent Inhalation Aerosol.

Rats and dogs survived the maximum practicable inhalation doses of salmeterol of 2.9 and 0.7 mg/kg, respectively. The maximum nonlethal oral doses in mice and rats were approximately 150 mg/kg and >1,000 mg/kg, respectively.

Dialysis is not appropriate treatment for overdosage of Serevent Inhalation Aerosol.

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February 1994
RL-096

References:

1. Pearlman DS, Chervinsky P, LaForce C, et al. A comparison of salmeterol with albuterol in the treatment of mild-to-moderate asthma. N Engl J Med. *November 1992;327:1420-1425.* ***2.*** *Data on file, Glaxo Inc.* ***3.*** *Ullman A, Svedmyr N. Salmeterol, a new long acting inhaled β_2 adrenoceptor agonist: comparison with salbutamol in adult asthmatic patients.* Thorax. *1988;43:674-678.* ***4.*** *Britton MG, Earnshaw JS, Palmer JBD. A twelve month comparison of salmeterol with salbutamol in asthmatic patients.* Eur Respir J. *1992;5:1062-1067.*

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PANTS: Leadership members of the Society of Critical Care Medicine and the American Academy of Pediatrics. RESULTS: Members of the Society of Critical Care Medicine and the American Academy of Pediatrics met at annual meetings of the two organizations with the goal of updating guidelines for pediatric ICUs. It was agreed that pediatric intensive care is provided primarily at one level, although there exists a need for an additional level of care in geographically isolated areas to allow stabilization of children before transport and to avoid long-distance transfer for disorders of less complexity or low acuity. Guidelines were developed for (a) Levels I and II pediatric intensive care facilities and services; (b) hospital facilities and services; (c) personnel; (d) prehospital care and interhospital transport; (e) quality assurance; and (f) research and training. CONCLUSIONS: Use of these guidelines should assist (a) hospitals, in staffing and equipping these units; (b) physicians, as a reference in the referral and care of critically ill infants and children; (c) emergency medical services personnel, for proper prehospital triage; and (d) Levels I and II pediatric ICUs as a means of ensuring proper patient care and professional credibility.

A Comparison of Active Compression–Decompression Cardiopulmonary Resuscitation with Standard Cardiopulmonary Resuscitation for Cardiac Arrests Occurring in the Hospital—TJ Cohen, BG Goldner, PC Maccaro, AP Ardito, S Trazzera, MB Cohen, SR Dibs. N Engl J Med 1993;329:1918.

BACKGROUND: Recent studies have demonstrated improved cardiopulmonary circulation during cardiac arrest with the use of a hand-held suction device (Ambu CardioPump) to perform active compression-decompression cardiopulmonary resuscitation (CPR). The purpose of this study was to compare active compression-decompression with standard CPR during cardiac arrests in hospitalized patients. METHODS: All patients over the age of 18 years who had a witnessed cardiac arrest while hospitalized at our center were enrolled in this trial; they were randomly assigned according to their medical-record numbers to receive either active compression-decompression or standard CPR. The study end points were the rates of initial resuscitation, survival at 24 hours, hospital discharge, and neurologic outcome. Compressions were performed according to the recommendations of the American Heart Association (80 to 100 compressions per minute; depth of compression 3.8 to 5.1 cm [1.5 to 2 in]; and 50% of the cycle spent in compression). RESULTS: Sixty-two patients (45 men and 17 women) with a mean age (± SE) of 68 ± 2 years were entered into the trial. Sixty-two percent of the patients who underwent active compression-decompression were initially resuscitated, as compared with 30% of the patients who received standard CPR ($p < 0.03$); 45% of the patients who underwent active compression-decompression survived for at least 24 hours, as compared with 9% of patients who underwent standard CPR ($p < 0.004$). Two of the 62 study patients survived to hospital discharge; both were randomly assigned to receive active compression-decompression. Neurologic outcome, as measured by the Glasgow coma score, was better with active compression-decompression (8.0 ± 1.3) than with standard CPR (3.5 ± 0.3; $p < 0.02$). CONCLUSIONS: In this preliminary study, we found that, as compared with standard CPR, active compression-decompression CPR improved the rate of initial resuscitation, survival at 24 hours, and neurologic outcome after in-hospital cardiac arrest. Larger trials will be required to assess the potential benefit in terms of long-term survival.

A Tobacco-Specific Lung Carcinogen in the Urine of Men Exposed to Cigarette Smoke—SS Hecht, SG Carmella, SE Murphy, S Akerkar, KD Brunnemann, D Hoffmann. N Engl J Med 1993;329:1543.

BACKGROUND: Environmental tobacco smoke has been classified by the Environmental Protection Agency as a carcinogen causally associated with lung cancer in adults, but there have been no reports of lung carcinogens or their metabolites in the body fluids or tissues of nonsmokers exposed to environmental tobacco smoke. METHODS: Five male nonsmokers were exposed to sidestream cigarette smoke generated by machine smoking of reference cigarettes for 180 minutes on each of 2 days, 6 months apart. Sidestream smoke is the smoke that originates from the smoldering end of a cigarette between puffs. Twenty-four-hour urine samples were collected before and after exposure. The urine samples were analyzed for 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and its glucuronide, which are metabolites of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), a powerful lung carcinogen in rodents. NNAL is also a lung carcinogen in rodents. RESULTS: The urinary excretion of the metabolites increased after exposure to sidestream smoke in all the men. The mean (± SD) amount of NNAL and NNAL glucuronide was significantly higher after exposure than at baseline (33.9 ± 20.0 vs 8.4 ± 11.2 ng per 24 hours [127 ± 74 vs 31 ± 41 pmol per day], $p < 0.001$) and was correlated with urinary cotinine excretion ($r = 0.89$, $p < 0.001$). The nicotine concentrations in the air to which the men were exposed were comparable to those in a heavily smoke-polluted bar. CONCLU-

SIONS: Nonsmokers exposed to sidestream cigarette smoke take up and metabolize a lung carcinogen, which provides experimental support for the proposal that environmental tobacco smoke can cause lung cancer.

Depression following Myocardial Infarction: Impact on 6-Month Survival—N Frasure-Smith, F Lespérance, M Talajic. JAMA 1993; 270:1819.

OBJECTIVE: To determine if the diagnosis of major depression in patients hospitalized following myocardial infarction (MI) would have an independent impact on cardiac mortality over the first 6 months after discharge. DESIGN: Prospective evaluation of the impact of depression assessed using a modified version of the National Institute of Mental Health Diagnostic Interview Schedule for major depressive episode. Cox proportional hazards regression was used to evaluate the independent impact of depression after control for significant clinical predictors in the data set. SETTING: A large, university-affiliated hospital specializing in cardiac care, located in Montreal, Quebec. PATIENTS: All consenting patients (n = 222) who met established criteria for MI between August 1991 and July 1992 and who survived to be discharged from the hospital. Patients were interviewed between 5 and 15 days following the MI and were followed up for 6 months. There were no age limits (range 24 to 88 years; mean 60 years). The sample was 78% male. PRIMARY OUTCOME MEASURE: Survival status at 6 months. RESULTS: By 6 months, 12 patients had died. All deaths were due to cardiac causes. Depression was a significant predictor of mortality (hazard ratio 5.74; 95% confidence interval 4.61-6.87; p = 0.0006). The impact of depression remained after control for left ventricular dysfunction (Killip class) and previous MI, the multivariate significant predictors of mortality in the data set (adjusted hazard ratio 4.29; 95% confidence interval 3.14-5.44; p = 0.013). CONCLUSION: Major depression in patients hospitalized following an MI is an independent risk factor for mortality at 6 months. Its impact is at least equivalent to that of left ventricular dysfunction (Killip class) and history of previous MI. Additional study is needed to deter-

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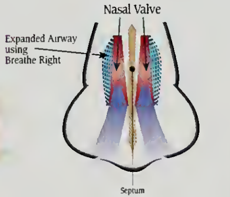
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"Deposition of aerosol from an MDI with a spacer or holding chamber is similar to (and perhaps better than) deposition from a properly used MDI alone."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

"In general, ... holding chamber) is the most convenient, versatile and cost-effective way to deliver aerosols."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

"MDIs can be used effectively in children. However, because of the inability of many pediatric patients younger than 10 years of age to coordinate the actuation of the MDI, a holding chamber should always be used. A holding chamber with mask should be used in those less than 3 years of age."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

" .... a holding chamber should be used with inhaled steroids for pediatric patients of any age."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

The Aerosol Consensus Statement of the AARC, the NIH expert panel and practitioners of respiratory care have established new *community standards* for the use of Metered Dose Inhaler aerosol treatments and therapy.

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"MDI holding chambers also eliminate the need to coordinate actuation and inhalation."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

"This study has demonstrated that a MDI plus aerosol holding chamber delivers a nearly fivefold greater dose of aerosolized drug to the lungs in comparison with a jet nebulizer in patients receiving mechanical ventilation."
H.D. Fuller, M.B. Dolovich, G. Posmituck, W. Wong Pack, and M.T. Newhouse. Pressurized Aerosol versus Jet Aerosol Delivery to Mechanically Ventilated Patients. Am Rev Respir Dis 1990; 141:440-444.

"The Aerochamber and MDI appears to be a highly efficient method of budesonide delivery to ventilated infants."
J. Grigg, et al Hammersmith Hospital, London, England. Delivery of therapeutic aerosols to intubated babies. Archives of Disease in Childhood 1992; 67: 25-30

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mine whether treatment of depression can influence post MI survival and to assess possible underlying mechanisms.

Sudden-Onset Fatal Asthma: A Distinct Entity with Few Eosinophils and Relatively More Neutrophils in the Airway Submucosa?—S Sur, TB Crotty, GM Kephart, BA Hyma, TV Colby, CE Reed, et al. Am Rev Respir Dis 1993;148:713.

To determine the histologic differences in the airways of patients who died from sudden-onset asthma and the more common slow-onset asthma, we studied seven cases of fatal asthma. The numbers of eosinophils and neutrophils, as well as extracellular deposition of their respective granule contents in the airway mucosa and submucosa, were determined and statistically analyzed. Four of the seven patients had slow-onset asthma attacks in which the time interval between onset of asthma and death was more than 2.5 h. In contrast, three patients had sudden-onset asthma in which the time interval between onset of asthma attack and death was less than 1 h. The four patients with slow-onset fatal asthma had more eosinophils (34.1 ± 6.3 in slow-onset; 9.7 ± 3.5 in sudden-onset; $p = 0.002$) and fewer neutrophils (4.8 ± 2.0 in slow-onset; 16.8 ± 5.4 in sudden-onset; $p = 0.008$) in the airway submucosa than did patients with sudden-onset fatal asthma. In addition, within the slow-onset fatal asthma group, eosinophils exceeded neutrophils in the airway submucosa (eosinophils > neutrophils, $p = 0.002$). By contrast, within the sudden-onset fatal asthma group, neutrophils exceeded eosinophils (neutrophils > eosinophils, $p = 0.04$). We suggest that sudden-onset fatal asthma is immunohistologically distinct from slow-onset fatal asthma and that it is characterized by a relative paucity of eosinophils in the face of an excess of neutrophils in the airway submucosa. These observations raise the possibility that the mechanism of airway inflammation as well as that of airway narrowing in sudden-onset fatal asthma may be quite distinct from those in slow-onset fatal asthma.

Asthma Control during and after Cessation of Regular Beta$_2$-Agonist Treatment—I Wahedna, CS Wong, AFZ Wisniewski, ID Pavord, AE Tattersfield. Am Rev Respir Dis 1993;148:707.

It has been suggested that regular treatment with high doses of β_2 agonists might result in poorer control of

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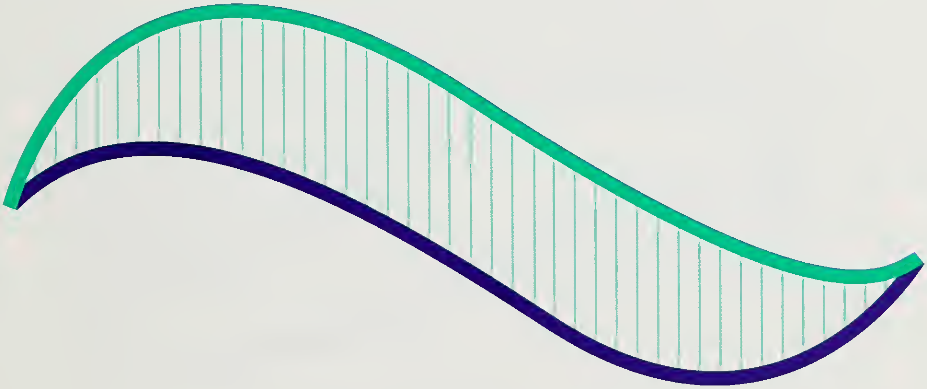

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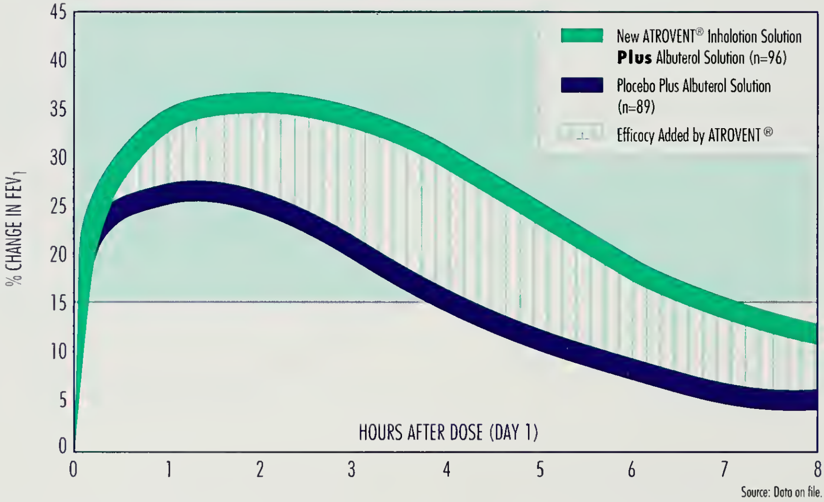

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In patients with COPD, ATROVENT Inhalation Solution plus albuterol solution produced significantly greater improvement in FEV_1 than albuterol alone.[1*]

ATROVENT Inhalation Solution is a bronchodilator for the maintenance treatment of bronchospasm in COPD.

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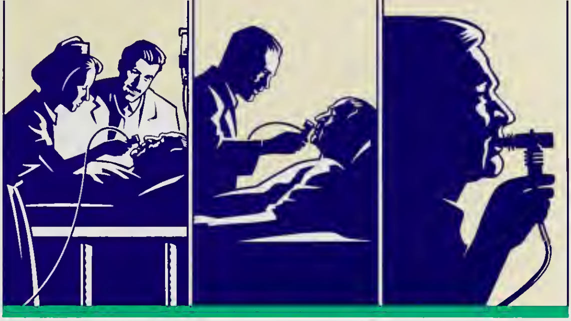
Please see brief summary of Prescribing Information on last page of this advertisement.



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The combination of ATROVENT Inhalation Solution and albuterol solution produced no clinically significant increase in side effects compared with albuterol alone.[2]

ATROVENT Inhalation Solution has a low incidence of systemic anticholinergic side effects.[3]

The use of ATROVENT Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation.

Combination of ATROVENT and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

ATROVENT should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy or bladder-neck obstruction.

Please see Prescribing Information for all adverse events, regardless of drug relationship, reported in the 12-week controlled trials of ATROVENT Inhalation Solution.

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Atrovent®
(ipratropium bromide)
Inhalation Solution

Brief Summary of Prescribing Information

CONTRAINDICATIONS Atrovent® (ipratropium bromide) is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives

WARNINGS The use of Atrovent® (ipratropium bromide) Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation. Combination of Atrovent and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation

PRECAUTIONS General Atrovent® (ipratropium bromide) should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder-neck obstruction

Information for Patients Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes. Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes Patients should be advised that Atrovent Inhalation Solution can be mixed in the nebulizer with albuterol if used within one hour Compatibility data are not currently available with other drugs Patients should be reminded that Atrovent Inhalation Solution should be used consistently as prescribed throughout the course of therapy

Drug Interactions Atrovent has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators. Atrovent has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions

Carcinogenesis, Mutagenesis, Impairment of Fertility Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of Atrovent Results of various mutagenicity studies (Ames test, mouse dominant lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by Atrovent administration At doses above 90 mg/kg, increased resorption and decreased conception rates were observed

Pregnancy *TERATOGENIC EFFECTS Pregnancy Category B* Oral reproduction studies performed in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively, and inhalation reproduction studies in rats and rabbits at doses of 1.5 and 1.8 mg/kg (or approximately 38 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of Atrovent However, no adequate or well-controlled studies have been conducted in pregnant women Because animal reproduction studies are not always predictive of human response, Atrovent should be used during pregnancy only if clearly needed

Nursing Mothers It is not known whether Atrovent is excreted in human milk Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that Atrovent® (ipratropium bromide) would reach the infant to a significant extent, especially when taken by inhalation, since Atrovent is not well absorbed systemically after inhalation or oral administration However, because many drugs are excreted in human milk, caution should be exercised when Atrovent is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children below the age of 12 have not been established

ADVERSE REACTIONS Adverse reaction information concerning Atrovent® (ipratropium bromide) Inhalation Solution is derived from 12-week active-controlled clinical trials Additional information is derived from foreign post-marketing experience and the published literature

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table below

All Adverse Events, from Double-blind, Parallel, 12-week Studies of Patients with COPD*

	PERCENT OF PATIENTS				
	Atrovent® (500 mcg t.i.d) n=219	**Alupent®** (15 mg t.i.d) n=212	**Atrovent®/Alupent®** (500 mcg t.i.d/ 15 mg t.i.d) n=108	**Albuterol** (2.5 mg t.i.d) n=205	**Atrovent®/Albuterol** (500 mcg t.i.d/ 2.5 mg t.i.d) n=100
Body as a Whole-General Disorders					
Headache	6.4	5.2	6.5	6.3	9.0
Pain	4.1	3.3	0.9	2.9	5.0
Influenza-like symptoms	3.7	4.7	6.5	0.5	1.0
Back pain	3.2	1.9	1.9	2.4	0.0
Chest pain	3.2	4.2	5.6	2.0	1.0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0.9	1.9	0.9	1.5	4.0
Central & Peripheral Nervous System					
Dizziness	2.3	3.3	1.9	3.9	4.0
Insomnia	0.9	0.5	4.6	1.0	1.0
Tremor	0.9	7.1	8.3	1.0	0.0
Nervousness	0.5	4.7	6.5	1.0	1.0
Gastrointestinal System Disorders					
Mouth Dryness	3.2	0.0	1.9	2.0	3.0
Nausea	4.1	3.8	1.9	2.9	2.0
Constipation	0.9	0.0	3.7	1.0	1.0
Musculo-skeletal System Disorders					
Arthritis	0.9	1.4	0.9	0.5	3.0
Respiratory System Disorders (Lower)					
Coughing	4.6	8.0	6.5	5.4	6.0
Dyspnea	9.6	13.2	16.7	12.7	9.0
Bronchitis	14.6	24.5	15.7	16.6	20.0
Bronchospasm	2.3	2.8	4.6	5.4	5.0
Sputum Increased	1.4	1.4	4.6	3.4	0.0
Respiratory Disorder	0.0	6.1	6.5	2.0	4.0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13.2	11.3	9.3	12.2	16.0
Pharyngitis	3.7	4.2	5.6	2.9	4.0
Rhinitis	2.3	4.2	1.9	2.4	0.0
Sinusitis	2.3	2.8	0.9	5.4	4.0

*All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with Atrovent include tachycardia, palpitations, eye pain, urinary retention, urinary tract infection and urticaria A single case of anaphylaxis thought to be possibly related to Atrovent has been reported. Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain have been reported

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most common events leading to discontinuation of Atrovent therapy in the 12-week trials Headache, mouth dryness and aggravation of COPD symptoms are more common when the total daily dose of Atrovent equals or exceeds 2,000 mcg

HOW SUPPLIED Atrovent® (ipratropium bromide) Inhalation Solution Unit-Dose Vial is supplied as a 0.02% clear, colorless solution containing 2.5 ml with 25 vials per foil pouch (NDC 0597-0080-62) Each vial is made from a low density polyethylene (LDPE) resin

Consult package insert before prescribing.

AS-8S-9/93

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* When used in combination with a beta-agonist solution, ATROVENT does not significantly increase side effects associated with the beta agonist alone.

References: **1.** Levin DC, Little KS, Laughlin KR, Galbraith JM. Ipratropium bromide solution in COPD augments extent and duration of FEV_1 increases achieved by albuterol up to 85 days. *Chest* 1993;104(suppl to No 2):112S. Abstract **2.** Data on file, Boehringer Ingelheim Pharmaceuticals, Inc. **3.** ATROVENT® (ipratropium bromide) Inhalation Solution full Prescribing Information.

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AS-5381

asthma and increased bronchial responsiveness. We have examined change in FEV_1 (ΔFEV_1), bronchial reactivity, peak expiratory flow (PEF), and symptoms during and after 3 wk of regular treatment with a relatively low dose of albuterol and broxaterol, a new β_2 agonist. Eleven subjects 18 to 50 yr of age with mild asthma inhaled albuterol (200 μg), broxaterol (400 μg), or placebo three times a day for 3 wk with a 2- to 4-wk run-in/washout period between treatments. Ipratropium bromide was allowed for symptomatic relief. The PD_{20} (dose of histamine causing a 20% fall in FEV_1) was measured before and 11, 35, and 59 h after cessation of treatment and a bronchodilator dose-response study before and 83 h after cessation of treatment. Change from baseline after albuterol and broxaterol are compared with change after placebo. Diurnal change in PEF (amplitude % mean) increased during treatment with albuterol by 6.5% (95% CI 1.7-12.3; $p < 0.02$) mainly because of a fall in morning PEF. Cessation of treatment with both $beta_2$ agonists was associated with a fall in FEV_1 and PD_{20} compared with placebo. After 11 h, mean FEV_1 had fallen by 10% after albuterol (95% CI 4.5-15.5; $p < 0.01$) and by 5.6% after broxaterol (95% CI 0.15-11.1; $p < 0.05$) compared with change after placebo; the greatest difference in ΔPD_{20} occurred 59 h after stopping treatment and was 1.47 doubling doses for albuterol (95% CI 0.6-2.4; $p < 0.01$) and 0.95 doubling doses for broxaterol (95% CI 0.05-1.8; $p < 0.05$). There was no significant change in the bronchodilator dose-response curves after either β_2 agonist. Thus, regular treatment with albuterol 200 μg three times a day for 3 wk was associated with an increase in PEF variability and, after cessation of treatment, a fall in FEV_1 and increased bronchial reactivity that persisted for 59 h. We conclude that adverse effects can occur both during and after cessation of 3 wk of regular treatment with relatively low doses of β_2 agonists.

Performance of an Asthma Quality of Life Questionnaire in an Outpatient Setting—BH Rowe, AD Oxman. Am Rev Respir Dis 1993; 148:675.

The objectives of this study were to assess the validity, reliability, and responsiveness of a disease-specific quality of life questionnaire (AQLQ) in asthma patients presenting for assessment and treatment in an emergency department (ED). Fifty-two patients 18 to 64 yrs of age were surveyed in three EDs. Admitted and discharged patients with stable and unstable asthma were included. Patients were interviewed prior to discharge from the ED. They completed a self-report asthma questionnaire, the self-administered Sickness Impact Profile (SIP) and AQLQ, a global assessment of asthma severity, a symptom questionnaire, and pulmonary function testing. Forty-three (83%) patients were available for follow-up at 7 to 10 days, and they completed a similar series of tests. Moderate correlations were found between total SIP and AQLQ measurements ($r = 0.49$; $p < 0.001$). Correlations were high between physical domain scores of the SIP and AQLQ symptoms ($r = 0.58$; $p < 0.0001$) and activity limitations ($r = 0.50$; $p < 0.0001$). Low correlations were found between pulmonary function results and AQLQ domains, except activity limitations ($r = 0.44$; $p < 0.001$). High correlations were found between symptoms or global assessments and AQLQ ($r > 0.6$; $p < 0.0001$). The instrument is highly responsive to small changes in patient status and outperforms pulmonary function tests and symptom scores in this setting. Test-retest reliability in patients who were stable was substantial (intraclass correlation coefficients > 0.9) on all aspects of the AQLQ. The AQLQ has been shown to be a reliable, valid, and responsive instrument for use in outpatient studies of patients who present with exacerbations of asthma and are discharged from the ED setting. Its use should be encouraged in future studies of asthmatic patients.

Asthma Risk and Occupation as a Respiratory Therapist—DC Christiani, DG Kern. Am Rev Respir Dis 1993;148:671.

In the modern hospital environment, many healthcare workers are exposed to hazardous substances. Among these hazards are respiratory sensitizers, irritants, and infectious agents. A previous cross-sectional study of Rhode Island respiratory therapists reported an excess risk of asthma after entry into that profession. Before the results of that study were published, we conducted a confirmatory mailed questionnaire survey of 2,086 Massachusetts respiratory therapists and 2,030 physical therapists and physical therapy assistants. Neither the survey questionnaire nor the accompanying cover letter revealed the focus of our investigation. A history of physician-diagnosed asthma was reported by 16% of respiratory therapists and 8% of control subjects. When analysis was restricted to those who developed asthma after entry into their profession, respiratory therapists still had a significant excess, 7.4 vs 2.8%. The odds ratio for respiratory therapy was 2.5 (95% CI 1.6-3.3) after adjustment for age, family history, atopic history, smoking, and gender. These results confirm the previous report of excess risk of asthma among respiratory therapists. This excess risk develops after entry into the profession and does not appear to be explained by bias or confounding. Efforts should be directed to identifying potential

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agents responsible for this form of occupational asthma.

Is the Association between Inhaled Beta-Agonist Use and Life-Threatening Asthma Because of Confounding by Severity?—P Ernst, B Habbick, S Suissa, B Hemmelgarn, D Cockcroft, AS Buist, et al. Am Rev Respir Dis 1993;148:75.

We have previously reported an increasing dose-response relationship between the regular use of beta-agonist inhalers and the risk of asthma death and near death among a cohort of 12,301 subjects who had been dispensed 10 or more prescriptions of asthma drugs from January 1980 to April 1987. That analysis was based solely on information obtained from linkable computerized databases. Such an association might be explained in part by the tendency of patients with more severe asthma, that is, those at greatest risk for an adverse outcome, to use more beta-agonist medication. To further examine this potential confounding by severity, we gathered clinical information independently from the field on the 129 case patients and their 655 control patients from the matched case-control analysis of 12,301 subjects. In 68% of the control patients with a life-threatening episode and 75% of the matched control subjects, we obtained a valid questionnaire from at least one physician who had seen the patient during the previous 2 yr. Acceptable information on hospitalizations because of asthma was obtained in 87% of those hospitalized. Clinical features associated with an increased risk of fatal and near-fatal asthma were a history of loss of consciousness or seizures during a previous asthma attack (odds ratio 10.2; 95% CI 3.9-26.7), a history of attacks of asthma precipitated by eating certain foods (odds ratio 5.1; 95% CI 2.4-11.1), a clinical score designed to reflect the severity of prior attacks of asthma leading to hospitalization, and prior respiratory acidosis among those in whom a blood gas determination was recorded. After adjustment for these clinical markers of asthma severity, there was no significant reduction in the odds ratio for asthma death and near death per metered-dose inhaler of fenoterol (eg, odds ratio 2.5; 95% CI 1.7-3.8) or albuterol (eg, odds ratio, 2.0; 95% CI 1.5- 2.7). We conclude that this further attempt at adjusting for confounding by severity did not alter the relationship between the use of beta-agonist inhalers by subjects outside of hospital and the risk of asthma death and near death.

Fast Versus Slow Ventilation for Neonates—S Kano, CJ Lanteri, PJ Pemberton, PN Lesouef, PD Sly. Am Rev Respir Dis 1993;148:578.

To investigate the effect of ventilation rate on respiratory mechanics, 21 neonates ventilated in the neonatal period for various reasons were studied while being ventilated at 30 and 80 breaths/min. Dynamic respiratory system elasticity (E_{RS}), dynamic respiratory system resistance (R_{RS}), and alveolar pressure at end expiration (EEP) were calculated by using multilinear regression to fit the equation of motion of a linear single-compartment model. Technically satisfactory data were obtained from 13 neonates. With the fast ventilation rate, tidal volume and R_{RS} decreased by a mean of 41.3% ($p < 0.01$) and 17.5% ($p < 0.01$), respectively. E_{RS} and EEP increased by a mean of 8.3% ($p < 0.05$) and 22.2% ($p < 0.01$), respectively. Fast ventilation produced a shorter effective time constant during expiration, limiting the changes in EEP and, hence, in end-expiratory lung volume. The same changes in respiratory mechanics were also observed in neonates who did not show an increase in EEP even at high frequency. These neonates had a high elastance and time constant short enough to ensure adequate lung emptying. These results suggest that the respiratory mechanics of ventilated neonates are frequency dependent and that neonates with higher E_{RS}, such as those with hyaline membrane disease, can cope with fast rate ventilation without developing dynamic hyperinflation.

Sleep Apnea and Cardiac Arrhythmias: Is There a Relationship?—WW Flemons, JE Remmers, AM Gillis. Am Rev Respir Dis 1993; 148:618.

To determine if there was a relationship between cardiac arrhyhmias and sleep apnea, we studied the prevalence of arrhythmias in a consecutive series of patients referred to our clinic for assessment of this disorder. Two hundred of 263 physician-referred patients were eligible for the study and, of these, 173 (86.5%) had complete investigations. All patients underwent a full night of polysomnography and Holter monitoring. Sleep apnea was diagnosed if patients had more than 10 apneas and hypopneas per hour (AHI). In 76 patients (43.9%) sleep apnea was diagnosed (median AHI = 33). The prevalence of arrhythmias in patients with sleep apnea versus those without was, respectively, complex ventricular ectopy (including ventricular tachycardia), 1.3% (95% CI 0.4-6.9) vs 4.1% (CI 1.6-10.1); frequent ventricular premature beats (> 30/h), 2.6% (CI 0.8-8.9) vs 6.2% (CI 2.9-12.8); second-degree atrioventricular block, 1.3% (CI 0.4-6.9) vs 4.1% (CI 1.6-10.1); sinus arrest, 5.2% (CI 2.2-12.6) vs 1.0% (CI 0.2-5.6). None of these differences was statistically significant. We conclude that the prevalence of cardiac arrhythmias is low in patients without serious cardiac or respiratory comorbidity who are referred for assessment of sleep apnea. Furthermore, the presence or absence of arrhythmias in this group is unrelated to sleep apnea severity.

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which covers the cost of printing and postage.

Tidal Volume Measurements in Newborns Using Respiratory Inductive Plethysmography—JA Adams, IA Zabaleta, D Stroh, P Johnson, MA Sackner. Am Rev Respir Dis 1993;148:585.

Respiratory inductive plethysmography (RIP) is a well-accepted noninvasive technology for monitoring breathing patterns in adults. Prior attempts to calibrate this device in babies have been fraught with technical difficulties, thereby limiting applications in this population. Recently, a new method, qualitative diagnostic calibration (QDC), has been shown to provide accurate calibration of tidal volume in adults. The QDC method is based upon principles of the isovolume maneuver and carried out during natural breathing without specialized respiratory maneuvers or postural changes. We calibrated RIP with QDC in the supine posture and compared tidal volume (V_T) measured with RIP to V_T by a face mask-pneumotachograph (PNT) in 21 healthy full-term newborns in supine and prone postures. Eleven of the babies were calibrated during active sleep and 10 in quiet sleep. The mean V_T in the supine and prone postures were 19 and 25 mL, respectively. In the supine and prone postures, weighted mean difference between RIP (V_T) and PNT (V_T) and 95% confidence intervals were –0.05 mL (–0.27, 0.18) and –0.32 mL (–0.08, 0.55), respectively. There was no difference in the accuracy of RIP relative to PNT calibrated during active sleep when thoracoabdominal incoordination was present or quiet sleep when it was not in either the supine or the prone postures. Therefore, in full-term infants, RIP calibrated with QDC solely in the supine posture provides clinically acceptable measurements of V_T in both supine and prone postures.

Predictors of Asthma and Wheezing in Adults: Grain Farming, Sex, and Smoking—A Senthilselvan, Y Chen, JA Dosman. Am Rev Respir Dis 1993;148:667.

We investigated predictors for asthma and wheeze in 1,634 men and women in the age group 20 to 65 yr from the town of Humboldt, Saskatchewan. On the basis of questionnaire responses, subjects were classified into mutually exclusive groups as asthmatic (n = 62), wheezing (n = 444), asymptomatic (n = 908), and symptomatic (n = 220) groups. After excluding the symptomatic group, we used polytomous logistic regression models to determine predictors of asthma and wheezing. Significant predictors for asthma were grain farming (odds ratio [OR] = 1.9, 95% confidence interval [CI] 1.1-3.5; $p = 0.03$) and sex (OR = 1.9, CI 1.1-3.2; $p = 0.03$; males compared with females). Significant predictors for wheezing were smoking (former smoker: OR = 1.8, CI 1.3-2.5, $p < 0.001$; current smoker: OR = 5.0, CI 3.8-6.7, $p < 0.001$; in comparison to nonsmoker) and grain farming (OR = 1.7, CI 1.3-2.4, $p < 0.001$). Age, level of education, and physical activity at work were not significant predictors for asthma or wheezing. None of the interaction effects between the predictors was significant. When stratified by sex, grain farming was a significant predictor of asthma in men but not in women. Nevertheless, smoking and grain farming were significant predictors of wheezing in both men and women. Our study raises the possibility that grain farming might be a risk factor for asthma and asthma-like symptoms.

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Optimizing Alveolar Expansion Prolongs the Effectiveness of Exogenous Surfactant Therapy in the Adult Rabbit—AB Froese, PR McCulloch, M Sugiura, S Vaclavik, F Possmayer, F Moller. Am Rev Respir Dis 1993;148:569.

We evaluated four ventilator patterns after the administration of 80 mg/kg bovine lipid extract surfactant (LES) to anesthetized, paralyzed, saline-lavaged New Zealand white rabbits. Two ventilator types were compared: high frequency oscillatory ventilation (HFO) versus conventional mechanical ventilation (CMV), each at high (HI) and low (LO) end-expiratory lung volumes (EELV); n = 6, each group; treatment duration = 4 h. Target P_{aO_2} ranges were > 350 mm Hg for groups with high EELV (ie, HFO-HI and CMV-HI) and 70 to 100 mm Hg for those with low EELV (ie, HFO-LO and CMV-LO). Ventilator pressures were limited to ≤ 39/9 cm H_2O in the CMV-HI group. Five of six CMV-HI-treated animals did not maintain target P_{aO_2} levels. Both ventilator type and strategy influenced outcome significantly. Animals managed with HFO had higher mean arterial pressures ($p = 0.004$), lower mean airway pressures ($\bar{P}_{aw}$) ($p < 0.00008$) and HCO_3^- requirements ($p < 0.02$), larger inflation ($p = 0.003$) and deflation ($p < 0.00001$) respiratory system volumes at 10 cm inflation pressure, and higher lung lamellar body ($p = 0.0006$) and lavage fluid ($p = 0.003$) phospholipid quantities than did CMV-treated animals. The deflation P-V curve ($p = 0.0004$), lamellar body ($p < 0.00001$) and lavage fluid ($p = 0.0002$) phospholipid levels were superior after the high EELV strategy. We conclude that ventilator pattern strongly influences exogenous surfactant efficacy. Benefits arise from keeping EELV high enough to prevent atelectasis and using small (≈ 2 mL/kg) tidal volumes to prevent over distension. Such a pattern was readily achieved with HFO using volume recruitment maneuvers to get the lung onto the deflation limb of its pressure-volume relationship followed by small volume cycles at high rates and sufficient mean pressure to maintain alveolar volume above the point of airway/alveolar closure.

Race and Gender Differences in Respiratory Illness Prevalence and Their Relationship to Environmental Exposures in Children 7 to 14 Years of Age—DR Gold, A Rotnitzky, AI Damokosh, JH Ware, FE Speizer, BG Ferris Jr, DW Dockery. Am Rev Respir Dis 1993; 148:10.

Race and gender differences in respiratory illness prevalence rates were assessed in a cohort of 8,322 White children and 1,056 Black children 7 to 14 yr of age from four U.S. cities. Boys had higher rates of wheeze, asthma, cough, phlegm, and bronchitis than girls. Black children had higher rates of persistent wheeze, shortness of breath with wheeze, asthma, chronic cough, and chronic phlegm than White children. We examined whether the racial disparity in respiratory illness prevalence could be accounted for by environmental exposures and socioeconomic factors. The proportion of families without a parent who had graduated from high school was higher for Blacks than for Whites, as was the proportion of single-parent households. Black children took up smoking less frequently; their mothers smoked fewer cigarettes. Personal and maternal smoking predicted higher rates of persistent wheeze, chronic cough, chronic phlegm, and chest illness. The relative odds for persistent wheeze were 1.34 (1.07, 1.69) for smoking children compared with nonsmoking children. The relative odds for persistent wheeze were 1.35 (1.13, 1.60) for children whose mother smoked > 30 cigarettes per day versus children with no maternal smoke exposure. Other predictors of respiratory illnesses included parental respiratory illness, parental education, only-child status, single-parent household, air conditioner use, and body mass index. Nevertheless, adjustment for socioeconomic factors, environmental exposures, and body habitus did not significantly reduce the excess respiratory illness prevalence observed among Black children. The adjusted relative odds were 1.47 (1.25, 1.74) for persistent wheeze and 1.57 (1.17, 2.10) for asthma for Black children versus White children. Population differences in unmeasured environmental exposures or in predisposition to allergy may account for residual differences between Black and White children in respiratory illness rates.

Alterations in Pulmonary Surfactant Composition and Activity after Experimental Lung Transplantation—RAW Veldhuizen, J Lee, D Sandler, W Hull, JA Whitsett, J Lewis, et al. Am Rev Respir Dis 1993;148:208.

Pulmonary surfactant facilitates breathing by reducing the surface tension at the air-liquid interface. We examined the effect of experimental lung transplantation on the phospholipid pool sizes of alveolar surfactant large and small aggregates, the composition of the large aggregates, the surface tension-reducing ability of lipid extract surfactant, and the leakage of serum proteins into the lung. A double-lung block from the donor animal was stored for 2 or 12 h after perfusion with either Euro-Collins solution or University of Wisconsin solution. The right donor lung was lavaged immediately after the storage period to determine the effects of storage on pulmonary surfactant. The left donor lung was transplanted. The recipient animal, containing its own

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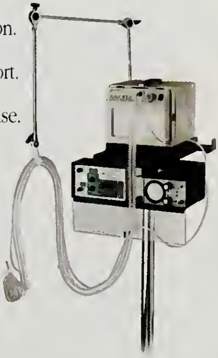

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native right lung and the transplanted left lung, was reperfused for 6 h. After the reperfusion period, the transplanted left lung and the native right lung were lavaged. After an ischemic time of 12 h, impaired gas exchange was observed in the transplanted lung as well as the native lung during the 6 h of reperfusion. This impaired gas exchange was associated with several significant changes in pulmonary surfactant: (1) total serum protein in the lung lavage was increased, (2) the small to large surfactant aggregate ratio was increased, (3) sphingomyelin content was increased and phosphatidylglycerol content was decreased in large aggregates, and (4) the surfactant-associated protein A content was decreased in large aggregates. No significant differences were observed between the results obtained with Euro-Collins and University of Wisconsin solutions. We conclude that prolonged storage of the donor lung before transplantation results in ischemic damage that produces an increase in potential surfactant inhibitors and can alter the small to large surfactant aggregate ratio after lung transplantation. These alterations in pulmonary surfactant resemble those observed in many adult respiratory distress syndrome types of injuries. We conclude that surfactant supplementation in lung transplantation should be investigated.

Efficacy and Safety of Short-Term Administration of Aerosolized Recombinant Human Deoxyribonuclease in Patients with Cystic Fibrosis—BW Ramsey, SJ Astley, ML Aitken, W Burke, AA Colin, HL Dorkin, et al. Am Rev Respir Dis 1993;148:145.

Chronic endobronchial bacterial infection evokes purulent airway secretions in patients with CF. The viscoelastic properties of these secretions is primarily due to the presence of polymerized DNA from degenerating leukocytes. Recombinant human DNase I (rhDNase) reduces the viscosity of CF sputum in vitro. To test the hypothesis that rhDNase would improve pulmonary function in children and adults with CF, we compared the efficacy and safety of 10-day administration of three doses of aerosolized rhDNase (0.6, 2.5, or 10.0 mg twice daily) in 181 outpatients using a randomized, placebo-controlled parallel design. Forced vital capacity (FVC) improved 10 to 12% ($p < 0.05$ to 0.001), and forced expiratory volume in one second (FEV_1) improved 10 to 15% ($p < 0.001$) across all doses of rhDNase compared with placebo. The magnitude of effect was dose dependent for both FVC and FEV_1 through study Day 21 ($p < 0.001$). rhDNase was associated with a decreased perception of dyspnea and an improved perception of well-being. No patients developed detectable anti-rhDNase antibodies or bronchial reactivity to rhDNase. Some patients experienced mild upper airway irritation, but no major adverse events were reported. Administration for 10 days of aerosolized rhDNase to pediatric and adult outpatients with CF improves lung function and is well tolerated. Although all three doses were efficacious, the greatest improvement in FEV_1 and FEV_1/FVC ratio was demonstrated in the 2.5 and 10.0 mg rhDNase treatment groups.

Interrupter Mechanics of Patients Admitted to a Chronic Ventilator Dependency Unit—MA Reinoso, DR Gracey, RD Hubmayr. Am Rev Respir Dis 1993;148:127.

We analyzed the results of interrupter mechanics tests of 73 consecutive patients who were admitted to a chronic ventilator dependency unit. The purposes of this study were (1) to establish guidelines for grading the severity of airway obstruction based on interrupter mechanics measurements; (2) to estimate the prevalence of airway obstruction in this population; (3) to compare interrupter test results with the clinical assessment of airway function; and (4) to characterize the distribution of bronchodilator-induced changes in isorecoil flow. Measurements from 65 of 73 patients (89%) were considered technically adequate. Of 65 patients, 29 (45%) showed signs of flow limitation during passive expiration. All patients with severe obstruction documented with spirometry during clinical stability were flow-limited and achieved maximal flows ≤ 0.45 L/s at recoil pressures of 10 cm H_2O. In contrast to measures of expiratory dynamics, the inspiratory resistance of the respiratory system was a poor index of severity of obstruction. In 7 of 12 patients, interrupter testing failed to substantiate a clinical diagnosis of severe obstruction while revealing unsuspected obstruction in 6 of 25 (24%) patients. The administration of 270 μg of albuterol increased flows at comparable recoil pressures by ≥ 0.1 L/s in 29% of 41 patients. Changes in flow were unimodally distributed and were not correlated with severity of obstruction. In summary, the routine use of the interrupter technique in ventilator-dependent patients is helpful in determining the severity and prevalence of airway obstruction, in identifying patients with otherwise unsuspected expiratory airflow limitation or those in whom this diagnosis is clinically suspected but cannot be substantiated, and in assessing the individual responses to bronchodilators.

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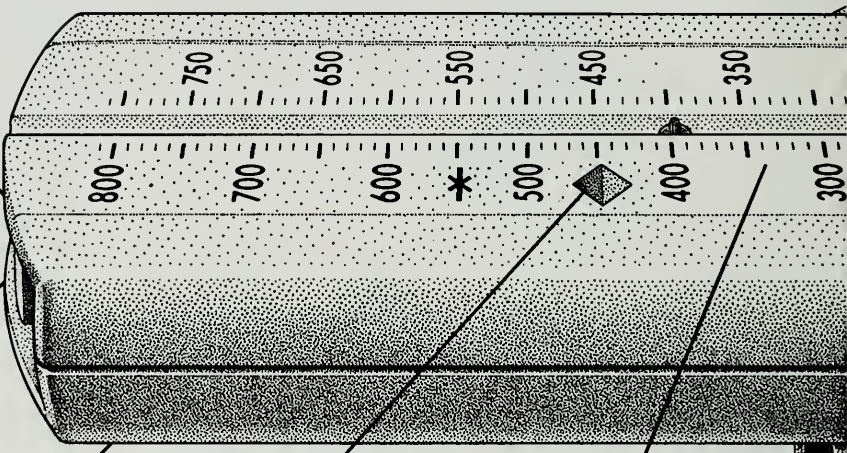

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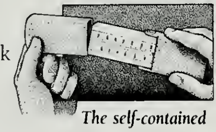

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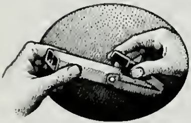

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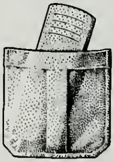

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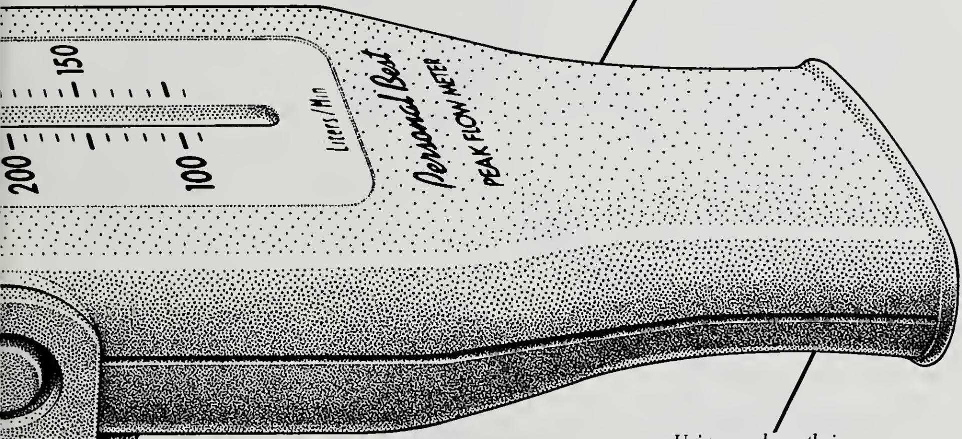

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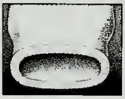

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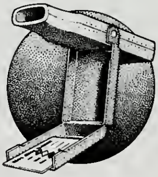

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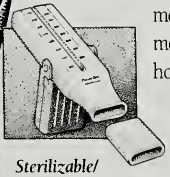

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Dear Reader:

The American Association for Respiratory Care and its science journal RESPIRATORY CARE are proud to present the April and May 1994 issues containing the proceedings from the 1993 Journal Conference "Controversies in Home Respiratory Care." The papers and discussions presented here are well-organized state-of-the-art materials designed for you to use in your daily practice.

Considerable planning and work are required to present and publish the proceedings of a conference like this. We are grateful to the speakers, the Editorial Board, Conference Co-Chairmen Robert Kacmarek and James K Stoller, Editor Pat Brougher, Associate Editor Kaye Weber, and Assistant Editor Donna Stephens for planning, writing, and editing these issues and for participating in the Conference and sharing this important information with us.

We are indebted to Allen & Hanburys, Division of Glaxo Inc, for their generous grant to the Association, which made the presentation and publication of the Conference proceedings possible. Allen & Hanburys has supported the last six Journal Conferences—for this we are enormously grateful.

Sincerely,

Ray Masferrer

Ray Masferrer RRT
Managing Editor
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A Review of Airway Clearance: New Techniques, Indications, and Recommendations

Karen Ann Hardy MD

- **I. Introduction**
- **II. Normal Clearance**
- **III. Abnormal Clearance**
- **IV. Diseases & Conditions associated with Abnormal Clearance**
- **V. Airway Clearance Techniques (ACT)**
  - **A. Cough**
  - **B. Postural Drainage, Percussion, & Vibration (PDPV)**
  - **C. Forced Exhalation Technique (FET)**
  - **D. Active Cycle of Breathing (ACB)**
  - **E. Positive Expiratory Pressure (PEP)**
  - **F. Autogenic Drainage (AD)**
  - **G. High Frequency Chest-Wall Compression (HFCC)**
  - **H. Flutter Valve Application**
  - **I. Exercise**
- **VI. Selection of Airway Clearance Technique**
- **VII. Summary & Recommendations**

Introduction

Bronchial hygiene therapies have been known most traditionally as chest physical therapy (CPT) and have been provided in the home for many decades. CPT usually refers to postural drainage, percussion, and vibration (PDPV) as described in standard textbooks of respiratory care.[1,2] A number of newer techniques have been described recently and increase the caregiver's decision dilemmas regarding therapy (newly dubbed airway clearance techniques, ACTs). Goals of treatment with all of these methods are to improve the clearance of airway secretions, thereby decreasing obstruction of the airways with the hope of improving the homogeneity of ventilation and gas exchange. An understanding of normal clearance and how certain diseases change the removal of secretions is essential. In this paper, I seek to integrate this information with the theoretical function of the various ACTs. In the final selection of a treatment, one must also consider cost and patient-specific and environmental components.[3] Ongoing evaluation of an initial treatment choice for effectiveness, efficiency, and adherence should be continuously challenged as the newer ACTs are more widely applied and studied.

Dr Hardy is Chief of Pulmonary & Cystic Fibrosis Center, California Pacific Medical Center, San Francisco, California.

The author has no financial interest in any of the products mentioned in the paper or in any competing products.

A version of this paper was presented during the RESPIRATORY CARE Journal Conference "Controversies in Home Respiratory Care," held in St Petersburg Beach, Florida, October 29-31, 1993.

Reprints: Karen Ann Hardy MD, Chief, Pediatric Pulmonary & Cystic Fibrosis Center, California Pacific Medical Center, San Francisco CA 94115.

Desirable features of any ACT relate to its effectiveness. In other words, in its clinical application, can it meet the goals of treatment without complications? Techniques should also be easy to teach to patients and should be easy for patients to perform independently or with assistance, depending on the patient's age and physical condition. Techniques should not fatigue the patient but rather be comfortable and time-efficient whenever possible. Cost-effective treatments that rely upon patient participation rather than expensive equipment are even more important in this age of managed health dollars. Techniques that can be completed in any setting and adapted for changing severity of illness are preferable. Finally, techniques that do depend on other individuals should be easy for caregivers to learn and be nonfatiguing to them as well. Many of these features of our current array of ACTs are known, but many others need to be clarified. Proponents of each technique strongly champion their method at symposia and national and international meetings. It is becoming clearer that other considerations such as cultural differences play a role in determining which method is appropriate for a given patient or caregiver. Controversy reigns in this field with many questions and few answers.

Normal Clearance

Normal clearance of the airways rests on two basic processes: mucociliary clearance and effective cough. Mucus is normally found throughout the respiratory tract, including lower airways, sinuses, and upper airways. The mucus layer is represented as an inferior sol layer that approximates and enmeshes with the cilia[4] and a gel layer above that effectively traps particulate matter and any microorganisms that might be inhaled and deposited (Fig. 1). Normal cilia are present on the luminal surface of pseudostratified columnar epithelial cells throughout the airways. Normal cilia are intrinsically active, beating in a unified direction to move the mucus layer toward the airway opening. The effective function of the cilia rests upon their unified beat, and research into the ultrastructure of the cilium has led to an increased understanding of dysfunctional cilia. Mucociliary clearance is effective in distal airways in which interaction between air and liquid cannot occur. The so-called mucociliary escalator brings secretions toward the large airways where effective cough causing air-liquid interaction becomes important to clearance of airway secretions.

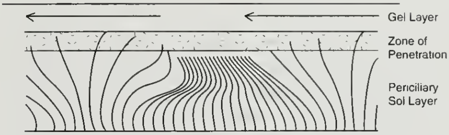

Fig. 1. Components of the mucociliary escalator.

An effective cough consists of three phases: an inspiratory gasp, a compressive phase, and an expulsive phase.[5] The reflexive inspiratory gasp facilitates an increase in lung volume to total lung capacity. Sudden closure of the glottis, especially related to false-cord closure and subsequent active expiratory muscle action, allows the patient to generate increased intrathoracic pressure. Sudden relaxation at the glottis initiates the expulsive phase, with subsequent dynamic change in the airway dimension downstream of the equal pressure point (ie, the point at which airway and pleural pressures are equal). Velocities of airflow in this area become high, up to three fourths of the speed of sound. This increases the gas-liquid interaction along central airways, generating mist flow and transporting the mucus toward the mouth either to be swallowed or expectorated. This flow-limiting segment of the downstream airway is an important mechanism for clearance of secretions, increasing the shear force to remove mucus along airway walls. Airflow and enhanced interaction between air and mucus layer are integral to any method that might be designed to increase clearance and are often unbalanced or lacking in diseases that impair clearance.

Abnormal Clearance

In abnormal situations, any portion of this clearance system may be dysfunctional and lead to mucus retention. Accumulations of mucus in the airway have immediate effects: either full airway obstruction with subsequent absorption of gas and consequent atelectasis or partial obstruction of the airway leading to a ball-valve phenomenon and contributing to air trapping and regional overdistention. Inhomogeneity of ventilation, which can affect gas exchange, is the obvious consequence of these two processes. Infection follows airway obstruction, combining the products of microorganisms (such as proteases and elastases) that provoke host-mediated inflammatory responses. Chemical mediators such as leukotrienes, proteases, and elastases destroy airway walls and generate unstable airways. These overcompliant (floppy) airways can then massively dilate, creating a vicious cycle of further abnormality in airway clearance. Ongoing inflammation damages the surface epithelium in the airway and decreases the number of ciliated cells, and mucus production in abnormal airways is often increased.[6]

Diseases and Conditions associated with Abnormal Airway Clearance

A number of diseases are associated with abnormal clearance, including primary diseases that affect the composition and production of mucus or cilial structure and function and those that change cough. In cystic fibrosis (CF), an inherited disease, the solute concentration in mucus is altered due to increased sodium reabsorption and decreased chloride secretion at the luminal surface of respiratory epithelium.[7] This leads to subsequent dehydration of the periciliary layer so that mucus is not appropriately transported. Primary cilial abnormalities, under the umbrella title of dyskinetic cilia syndromes, also contribute to abnormal airway clearance.[8] Patients with such conditions either have cilia that are motile but dyskinetic (do not beat in an appropriately coordinated fashion) or cilia that are immotile. Any of these defects contribute to ineffective or absent cilial beating, leading to loss of the mucociliary escalator.

Cough is the patient's remaining major clearance mechanism, but ultimately airway damage decreases the effectiveness of that mechanism as well. Bronchiectasis, a sequela of abnormal clearance, is an irreversible final common finding in both diseases. Bronchiectasis can also develop by other pathways. These can include severe asthma, infections such as tuberculosis or severe pneumonia (eg, from pertussis or measles), foreign-body aspiration with subsequent lung damage, chronic smoking with chronic obstructive diseases,[9] or, more rarely, processes such as obliterative bronchiolitis.[10] Infections causing severe bronchiolitis or airway damage can also lead to abnormal clearance, not necessarily associated with bronchiectasis but requiring consideration of airway clearance techniques. Asthma and chronic bronchitis are associated with inflammatory changes of the airway epithelium that lead to airway obstruction in addition to mucus hypersecretion. Bronchospasm is a disease process also associated with the need for assisted clearance. Two other conditions deserve consideration for treatment based on primary abnormality in cough. Pain associated with breathing secondary to trauma or surgery (especially in the abdominal cavity) is an important reason to consider assisted airway clearance. Mucociliary escalator function may be normal but without effective cough, mucus plugs, obstruction, and atelectasis can be attributed to the pain associated with inspiration. Musculoskeletal or neurologic abnormalities such as muscular dystrophy, spinal muscular atrophy, myasthenia, poliomyelitis, or cerebral palsy can be associated with poor bellows function. Pure restrictive defects result in the smaller vital capacities and lower functional residual capacities classically associated with inability to cough effectively—again initiating the pathophysiologic sequence just described.

Airway Clearance Techniques

The types of airway clearance techniques have increased rapidly and have been described in a number of international journals. Table 1[11-42] displays those most commonly considered this year (1993). Cough continues to head the list followed by PDPV. Active forms of airway clearance include the forced exhalation technique (FET), huff cough, and the active cycle of breathing (ACB). Positive expiratory pressure (PEP) treatments with either mask or mouthpiece and autogenic drainage (AD) are two popular European techniques that are becoming more widespread in the United States. The application of high frequency oscillations to the chest wall (HFCC) or at the airway opening, or mouth, (HFao) is a technique that requires added equipment but is potentially applicable for patients who need a passive therapy.[34-38] The flutter valve,[39,40] devised in Switzerland, is just beginning to be evaluated in the U.S. Exercise remains an important consideration in airway clearance. I briefly describe the method of application, theoretical physiology, pulmonary function changes, and benefits and prob-

Table 1. Studies and Reviews Describing Airway Clearance Techniques

Technique	First Author	Reference
Cough	Holinger	5
	Kamei	11
Forced exhalatory technique (FET)	Pryor	12
Postural drainage, percussion, and vibration (PDPV)	Frownfelter	1
	Murray	13
	Eid	14
	Zapetal	15
	Murphy	16
	Selsby	17
	Pryor	18
	Feldman	19
	Desmond	20
	Reisman	21
Active Cycle of Breathing (ACB)	Pryor	22
	Miller	23
Positive Expiratory Pressure (PEP)	Mahlmeister	24
	Andersen	25
	Andersen	26
	Mortensen	27
	Oberwaldner	28
Autogenic drainage (AD)	Chevaillir	29
	Kraemer	30
	Spence	31
	Schoni	32
	McIlwaine	33
High frequency chest-wall compression (HFCC)	Warwick	34
	Freitag	35
	King	36
	Rubin	37
	Ahrens	38
High frequency at airway opening (HFao)	King	36
Flutter valve	Hüls	39
	Lindemann	40
Exercise	Zach	41
	Zach	42

lems for each technique. Classification of methods based on independent performance by the patient or the need for assistance and for active or passive work by the patient has become blurred as more techniques are being used together. Many of the techniques have been developed and studied using patients with CF. CF patients are urged to adhere to regimens of vigorous therapy early in life, and, therefore, new ACT options are a welcome change.

Cough

I have already described the three phases of a normal (effective) cough, and the reader is referred to other comprehensive reviews of cough physiology.[5,11] The effect of flow-limiting segments and cough on mucociliary clearance was reported by Smaldone and colleagues in 1979.[43] They showed that cough increased deposition of ^{99m}Tc-labeled albumin at the flow-limiting airway segment and locally impaired mucociliary clearance in anesthetized, instrumented dogs. In the same year, Oldenburg et al[44] compared mucus clearance during quiet breathing, postural drainage, and cough in a group of clinically stable patients with chronic bronchitis. They also used ^{99m}Tc-labeled albumin to determine that cough accelerated total lung and peripheral clearance but that postural drainage without coughing did not change clearance. They emphasized the need for studies to control for coughing as new clearance methods are described. De Boeck and Zinman[45] compared changes in pulmonary mechanics after cough vs after PDPV in moderately ill patients with CF and found that after 1 hour, maximal expiratory flowrates at 50% and 25% of the vital capacity were significantly improved after cough. Lung volumes and flows at high lung volumes were not affected, and sputum volumes did not correlate with pulmonary function. They suggested that cough be recommended as an easy alternative to PDPV. These findings were supported in sicker patients by Bain et al in 1988.[46] Ineffective cough occurs when dynamic compression of a segment causes complete flow limitation and obstructs the transport of mucus cephalad. Ineffective coughing can also lead to fatigue and spasms of cough can lead to hypoxia.

Postural Drainage, Percussion, & Vibration

PDPV is a conventional therapy in the U.S., devised and popularized over the first half of this century.[1] Clinical trials as we now know them were not applied to this technique, now considered by many to be the gold standard for chest physical therapy. This method requires positioning the patient into various drainage positions that anatomically favor the gravity-directed movement of secretions toward the airway opening. Manual percussion (with the hand alone or the hand plus an adjunct like a handheld mask) or mechanical percussion at the chest wall is applied while the patient breathes quietly in the drainage position. The patient then is asked to breathe and exhale while vibrations are applied during the exhalation phase. Directed coughing is typically requested after percussion and vibration are completed in each drainage position. Drainage times per position vary greatly, and total treatment times also vary with different diseases and during periods of exacerbation. For severely abnormal patients such as those with cystic fibrosis, drainage times of at least 3 minutes up to 10 minutes per position typically are utilized. Vibration is difficult to apply manually and is often ignored in many settings. Thus, this technique may be applied in a nonstandard manner in the home and in the hospital.

The theory behind this method is the self-explanatory ketchup bottle analogy well described by Murray in 1979.[13] It is also thought that percussion generates flow transients in airways beneath a percussed segment that improve the gas-liquid interactions. Parts of this technique (such as drainage, percussion, or vibration alone) have been studied in various disease processes and were reviewed by Eid and co-workers in this journal in 1991.[14] As discussed in that article, a number of components of this therapy are controversial, and studies fail to show that the addition of percussion and vibration is uniformly helpful. Drainage itself can assist mucociliary clearance, as shown by pulmonary function testing and nuclear clearance studies. Pulmonary function changes with PDPV vary among patients; some patients actually experience hemoglobin desaturation and may suffer during application of this technique,[15-18] whereas others may benefit.[19-21] In my experience, PDPV can be particularly helpful in young babies who can be treated by parents or health professionals but who are unable to actively cooperate with other ACTs. Another useful facet of this technique is the ability to localize treatment to certain segments.

Difficulties with PDPV include the necessity for assistance during its administration that causes older children and youth to be less likely to adhere, given their drive for greater independence. Compliance with this therapy is often poor.[47] Patients may do well with this technique initially but then develop new complications, such as gastroesophageal reflux or severe obstruction, and find the positions intolerable. Caregivers performing PDPV can suffer from pain or musculoskeletal abnormalities such as carpal tunnel syndrome. Eid et al's paper[14] clearly discusses a number of these negative issues. Use of some devices including mechanical and ultrasonic percussors and mechanical vibrators can help to avoid the problems of dependence on others and caregiver discomfort or disease. The few studies that have compared manual and mechanical treatments show that they can be equivalent.[48] Mechanical devices may range in price from less than a hundred to thousands of dollars. The frequency of their output is variable and may not be ideal for clearance—in general, percussors may be too slow and vibrators too rapid.[36]

Forced Exhalation Technique

The possibility of modifying the patient's cough to facilitate airway clearance has been a topic of great interest. As discussed previously, patients who have marked airway instability can experience changes with coughing that result in complete collapse of the airway above the equal pressure point rather than the airway's becoming a flow-limiting segment that increases gas velocity. With full collapse of the airway, the patient cannot clear or expel mucus. Patients with cystic fibrosis, dyskinetic cilial syndromes, and bronchiectasis are those most likely to benefit from cough modification. The forced exhalation technique was first described in New Zealand (personal communication, Jennifer Pryor MD, 1993), and then championed by the British.[12,22] The FET consists of one or two huffs (forced expirations), from mid- to low-lung volume, followed by a period of relaxed, controlled diaphragmatic breathing. The forced expiration can be augmented by brisk adduction of the upper arms to self-compress the thorax.[12] FET changes the position of the equal pressure point in the airway thereby stabilizing the airway and improving clearance. This technique has been studied alone and is shown to be an adjunct to PDPV rather than a replacement.[21]

Active Cycle of Breathing Techniques

In 1992, Pryor et al[22] renamed FET the active cycle of breathing, or ACB. They did this to emphasize that the FET always couples breathing exercises with the huff cough. As the technique spread to other countries it often became known merely as a type of cough. ACB, described by Pryor, is based on the principles of increasing gas-liquid interactions as described earlier and the notion that patient participation during airway clearance is important. Three components—thoracic expansion exercises, 'breathing control' (ie, diaphragmatic breathing or breathing relaxation), and the FET—are combined in a set cycle:

1. Relaxation and breathing control
2. Three to four thoracic expansion exercises (may add percussion, vibration, chest compressions)
3. Relaxation and breathing control
4. Three to four thoracic expansion exercises (may add percussion, vibration, chest compressions)
5. Relaxation and breathing control
6. One to two forced expirations (huffs)
7. Relaxation and breathing control

The theory behind the ACB technique includes the benefit of the FET mentioned earlier and the improvement of aeration of alveoli, which decreases inhomogeneity and improves the driving pressure in alveoli behind blocked airways. To my knowledge, no studies evaluating ACB technique have been published in the U.S. A recent abstract[23] from England compared the ACB technique to autogenic drainage and found both methods well tolerated and comparable in effect on sputum weight recovered and pulmonary mechanics. The benefits of this technique are independence for the older patient and the absence of desaturation or physical compromise during its performance. No costly equipment is required. The ACB technique is not useful for infants and young children (< 2 years). Problems with the technique need to be clarified through controlled comparative trials.

Positive Expiratory Pressure

The PEP technique, recently reviewed in this journal by Mahlmeister et al,[24] was developed in Denmark and is now so widely applied there that PDPV is rarely utilized. The patient breathes in and out 5-20 times through a flow resistor, which creates a positive pressure in the airways during exhalation. The pressure generated can be monitored and adjusted with a manometer. Either low pressures (15-30 cm H_2O) or high pressures (60-80 cm H_2O) may be prescribed. Active exhalations with a ratio of inspiration to exhalation of 1:3 or 1:4 are suggested. After the series of breaths, the patient performs the FET. This cycle is repeated until the patient has expelled secretions, usually within 20 minutes or less. The positive pressure generated by the mask as it was initially popularized in Denmark and the mouthpiece utilized in the U.S., in theory, improves aeration to alveoli through collateral ventilation. PEP allows gas to equilibrate through the pores of Kohn, filling underaerated or nonaerated segments. When the patient then performs huff maneuvers, he is able to generate flow and to expel mucus that blocks airways. Figure 2 illustrates this concept. Andersen and colleagues[25,26] demonstrated these principles in cadaver lungs and theorized that additional improvement in homogeneity occurs by traction of neighboring alveoli. PEP itself and the FET contribute to airway stability during the entire treatment period. All of these mechanisms should contribute to improved homogeneity of ventilation, improved gas exchange, and improved airway clearance.

At least 38 studies comparing PEP to other techniques have been published (mostly in the European literature) and were recently reviewed.[24] Early studies concentrated on the beneficial effects of PEP in patients at risk for postoperative atelectasis. In subsequent years, studies on patients with cystic fibrosis became more common, and an additional important study by Mortensen and colleagues was published in 1991.[27] Initially they compared PD/FET to PEP/FET with controls, measuring whole-lung and regional clearance with radiolabeled aerosol, and found similar effects with both treatments superior to control and not correlated with sputum volume. In 1993, Falk, Mortensen, and colleagues[49] compared PEP/FET to FET alone with controls showing improved radioaerosol clearance with either active technique but with a greater advantage to PEP. The longest evaluation period was reported by Oberwaldner et al[28] who compared lung function in patients with cystic fibrosis before and during the use of high pressure PEP. They showed that decreased

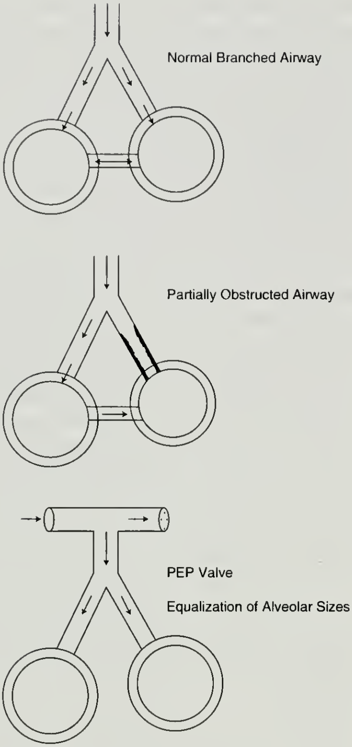

Fig. 2. Diagram illustrating probable forces active during positive expiratory pressure (PEP) therapy.

hyperinflation and improved lung function occurred with 10 months of PEP and that return to PDPV for only 2 months resulted in clinically important deterioration in function. Reinstitution of PEP for an additional 6 months resulted in return of lung function to the previous level.

PEP has not been reported to cause compromise with desaturation and can be performed independently. A particular advantage of this technique is its application in younger children—those 3 years and older are typically able to master the technique. Ongoing problems with this technique include the selection of an optimal PEP level, which is controversial, and the application in patients under 3 years of age. In my experience, the technique itself can be performed by severely ill patients and is helpful in conditions associated with atelectasis. Patients who have recently had facial surgery may find PEP too uncomfortable but can switch to another method during healing. Pneumothorax is a recognized potential complication of this technique and has been reported in a single patient in whom it developed during exercise, 4 hours after a treatment session using high pressure PEP.[30]

Autogenic Drainage

Autogenic drainage was devised in Belgium and is a controlled method of breathing that requires no added equipment. The patient is trained to breathe at three lung volumes, beginning at low-lung volume to 'unstick' the mucus, moving to mid-lung volume to 'collect' mucus, and then to high-volume maneuvers to 'expel' mucus.[29] Figure 3 is an example of a spirometric tracing from a patient going through a few breaths at each level. In all cases, the patients balance their maximal expiratory flows against collapse of dynamically unstable airways, trying to achieve a mucus rattle as opposed to a wheeze. Feedback including auditory, visual, and tactile sensing is important in teaching this technique.[30,31]

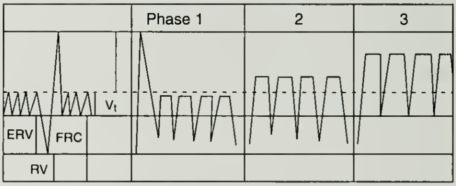

Fig. 3. Spirogram of lung volumes during phases of autogenic drainage. Phase 1—'unstick'; Phase 2—'collect'; Phase 3—evacuate. Subject had normal lung function.

The theory behind AD is to improve airflow in small airways so that the movement of mucus can be facilitated. Gas-liquid interactions are known to be necessary for airway clearance, and pulmonary function testing has shown that this method allows improved airflow at low lung volumes in comparison to airflows during cough and active breathing.[32] Figure 4 illustrates this principle, showing the changes in a flow-volume loop during maximal forced maneuvers and during AD breathing. To my knowledge, pulmonary function changes with this technique have been studied rarely, with only a single comparative study.[23] In a 1-year crossover trial with cystic fibrosis patients reported by McIlwaine et al in an abstract,[33] the actual clearance of sputum was markedly improved with this technique versus PEP or standard therapy.

Concerns have been raised about the difficulty of teaching this method, and our group has worked on biofeedback to improve visual input to the patient as an additional training method.[31] Increasing the patient's feedback during application of the technique does enhance learning the method, but in all cases multiple sessions are required to teach patients skillful application of AD. Patients are generally able to perform the AD technique with much less effort after they have practiced. Actual studies of energy expenditure during performance of this technique have not been published but will undoubtedly be pursued in the future. Teaching caregivers how to teach patients has also been discussed frequently in North America. In my experience, regular training and frequent practice with patients helps to keep the teacher's skills sharp. This technique is applicable to patients who are able to concentrate and are not so severely ill that they become fatigued. AD is performed independently and has the major benefit of not requiring any other equipment. Thus, it can be applied in a number of settings and during various activities of daily living.

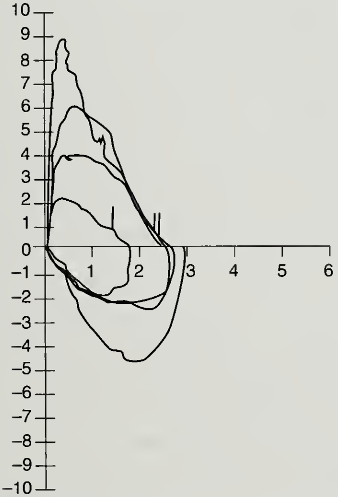

Fig. 4. Forced flow-volume loop and flow-volume loops performed by patient during phases of autogenic drainage at three lung volumes.

High Frequency Chest-Wall Compression

Methods for application of high frequency oscillations to the chest wall (ie, high frequency chest-wall compression, or HFCC) have been devised over the past 10 years by Warwick and colleagues[34] at the University of Minnesota. The ThAIRapy™ vest, or "the vest" as it is sometimes known, is an inflatable, personally fitted jacket attached to a large pump that generates variable, high frequency oscillations and applies them directly to the chest wall. In theory, these vibrations cause transient increases in airflow in airways, resulting in improved gas-liquid interactions and the movement of mucus. Animal studies[35,36] and clinical trials[37,38] have shown that the frequency of oscillations and the flow bias during treatment are important in determining ultimate effect of this type of treatment. Studies in anesthetized dogs have shown that if the flow bias during treatment is not expiratory, mucociliary clearance as measured by radionuclide studies may not improve, and mucus may actually move upstream (distally).[35,36] Additionally, the cycles/s (Hz) used in the application of energy to the chest wall are important both for patient comfort and efficacy.[37] Most recently, patients with cystic fibrosis who were admitted for acute pulmonary exacerbations were randomly treated with either the vest or conventional PDPV three times daily.[38] Weight gain, improvement in pulmonary function, and sputum mobilization were not significantly different in any of the 50 patients. Whitman et al reported similar experience in a 9-patient series.[50] Further studies are necessary. A benefit of such treatment would be the patient's freedom to pursue other activities while wearing the vest. As initially described, this is a passive method that can be altered by simultaneously performing other ACTs as described earlier. Older patients can attach themselves to this device independently and thus achieve airway clearance in the home or in other settings without assistance. To my knowledge, vests for infants and younger children are not now available (1993). A major detractor is the cost of the equipment, which rents for $500 a month and sells for about $20,000 (personal communication, Dale Klous, American Biosystems Inc, St Paul MN, 1993). New high frequency devices that apply oscil-

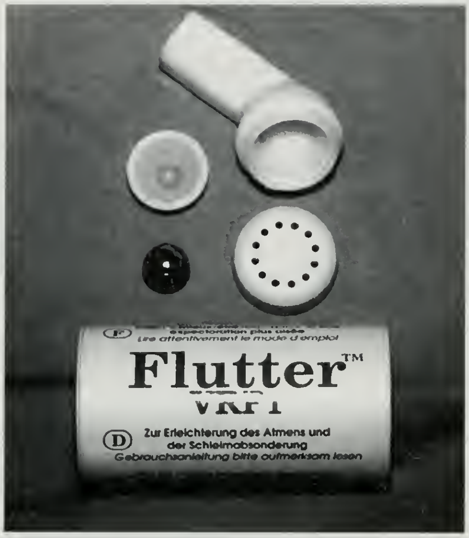
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Fig. 5. Flutter device designed to combine the techniques of positive expiratory pressure (PEP) and high frequency oscillation at the airway opening (HFao).

lations to the airway opening are being studied in patient and animal models but are not widely available for clinical use.

Flutter Valve Application

The flutter valve device was developed in Switzerland and combines the techniques of PEP with high frequency oscillations at the airway opening (HFao).[39,40] The device (Fig. 5) resembles a fat pipe with an inner cone leading to a loosely supported steel ball covered by a perforated cap. The patient exhales actively into the pipe, generating a positive expiratory pressure of 10-25 cm H_2O. During exhalation, the ball flutters in the pipe, generating oscillations of about 15 Hz, which are transmitted through the airways. The patient can control the pressure by changing the flowrate or the oscillations by changing the angle of the device in the mouth. The physiologic changes described for PEP and high frequency airway oscillations apply to the flutter. Pulmonary function measurements comparing the flutter and PEP on 8 cystic fibrosis patients in our laboratory showed no differences, although ongoing study of a larger group is needed to improve the power to show a difference between the two techniques if one exists. Sputum volume with the flutter compared to PDPV was significantly increased in 18 patients reported by Bolek[51] at the 7th Annual North American Cystic Fibrosis Conference, in Dallas, Texas in 1993.

Exercise

Exercise as an ACT offers many choices of activities that may appeal to all but the youngest patients. It improves overall aeration and ventilation-perfusion matching. It can increase sputum production and transiently improve pulmonary function.[41,42] This is the only ACT that has added advantages of improving general fitness, the patient's self-esteem, and measures of quality of life. Exercise is socially acceptable and encouraged for the healthy; thus, increased involvement helps to normalize the patient's life rather than adding a therapy that accentuates differences from peers. Exercise can be fatiguing and has been clearly associated with desaturation when a patient's pulmonary function is poor.[52] Further, infants and youngsters and patients with neuromuscular limitations are not eligible candidates.

Selection of Airway Clearance Technique

Selection criteria for these different techniques include consideration of a number of items (Table 2). Motivation is key to regular performance of any ACT. Many caregivers believe that any regimen applied faithfully helps patients maintain pulmonary function, but they also recognize that adherence to regimens is a major limitation. Repeating anything that does not cause an immediate change in one's feeling of health or well-being is always difficult. Sputum production, though frequently shown *not* to correlate with pulmonary function, is motivating. Most patients cannot help but believe that any ACT that helps them to produce large quantities of sputum when others do not, must be the treatment of choice. Sputum is the only immediate reinforcement that most of our patients have to encourage them to persist in a repetitive task.

The patient's age is important as is any preference for a particular technique. Patient and care-team goals for treatment should be discussed jointly with the intent of choosing the method that best fits

Table 2. Factors To Be Considered When Selecting an Airway Clearance Technique

Motivation
Patient's goals
Physician/caregiver goals
Effectiveness (of considered technique)
Patient's age
Patient's ability to concentrate
Ease (of learning and of teaching)
Skill of therapists/teachers
Fatigue or work required
Need for assistants or equipment
Limitations of technique based on disease type and severity
Costs (direct and indirect)
Desirability of combining methods

the patient's main concerns. The skills of the therapist available to teach techniques, in part, determine which ACT can be offered in a particular setting. Some institutions are able to provide qualified teachers for all methods, whereas others may need to concentrate on a few and teach those well. The ease with which the patient can perform the technique and with which the technique can be taught are important selection considerations. The fatigue experienced by the patient during any technique influences ACT selection and performance. I have observed that the efficacy of any one ACT in a given patient may be different from results of published studies and justifies providing the opportunity to try mutually agreed-upon strategies. If, after study by the patient, a number of methods appear equivalent, it makes sense to allow the patient to choose. This adds variety to the task while meeting treatment goals. The patient's disease may impose certain limitations; for example, patients with neuromuscular disease may not be able to cooperate adequately for some types of treatment. Today and in coming years, the cost of these techniques must be considered. If ongoing research proves that some ACTs are equivalent, the more expensive of the techniques are unjustified. Mechanical devices such as some percussors, the vest, and some oscillators for application at the airway opening are extremely expensive (personal communications, Dale Klous, American Biosystems Inc, and Alex Stensler, SensorMedics, 1993). ACB technique and AD require no additional equipment, and the PEP device is relatively inexpensive (personal communication, David Goodwin, DC Lung Co, 1993. *Editor's Note: DC Lung Co has been acquired by VP Mercury, Product Representative Gary Blount, 1-800-237-6418).* The flutter is not yet FDA-approved but is expected to be similar to PEP in cost.

Home care applications of ACT must consider all of the above. Additionally, the patient's need for support at home to help with some techniques must be considered. PDPV, as conventionally performed with an assistant, requires equipment for drainage positioning; and if family support is not available, an additional person must be brought into the home. The cost of this can be prohibitive, especially if multiple daily treatments are to be administered by a trained professional such as a respiratory care practitioner, physical therapist, or nurse. Mechanical devices to eliminate the need for additional help are expensive, may not be portable, and require the patient to be physically capable of assembling or utilizing the equipment. Occasionally, noise and inconvenience to roommates must be considered in choosing home therapy.

Summary and Recommendations

Airway clearance techniques are indicated for specific diseases that have known clearance abnormalities (Table 3). Murray[13] and others have commented that such techniques are required only for patients with a daily sputum production of ≥ 30 mL. I have observed that patients with diseases known to cause clearance abnormalities can have sputum clearance with some techniques such as PEP, AD, and ACB when PDPV has not been effective. It is therefore reasonable to consider airway clearance techniques for any patient who has a disease known to alter mucus clearance—including cystic fibrosis, dyskinetic cilial syndromes, and bronchiectasis from any cause. Patients with atelectasis from mucus plugs and hypersecretory states such as asthma and chronic bronchitis, patients with pain secondary to surgical procedures, and patients with neuromuscular disease may also benefit from the application of airway clearance techniques.

Infants and children up to about 3 years of age with airway clearance problems need to be treated with PDPV. Manual percussion with hands alone or a flexible face mask or cup and small mechanical vibrator/percussors, such as the ultrasonic devices, can be used. The high frequency oscillator is not

Table 3. Recommendations for Airway Clearance Techniques in Specific Conditions*

- Cystic fibrosis, cilial syndromes, bronchiectasis (require an ACT)
  - Infants—PDPV
  - 3-12 years—exercise, PEP, PDPV, ACB, HFCC (HFao or flutter valve)
  - > 12 years—exercise, ACB, AD, PEP, PDPV, HFCC
- Atelectasis
  - PEP, PDPV
- Prevention of postoperative atelectasis
  - PEP—Abdominal surgery, ACB
- Asthma (with mucus plugging but without airleak)
  - PEP, PDPV
- Neurologic abnormalities (spasticity, bulbar palsy, aspiration-prone)
  - PDPV, suction
- Musculoskeletal weakness (muscular dystrophy, myasthenia, poliomyelitis)
  - PEP, negative and positive-assist devices

*ACT = airway clearance technique; PDPV = postural drainage, percussion, and vibration; PEP = positive expiratory pressure; ACB = active cycle of breathing; HFCC = high frequency chest-wall compression; HFao = high frequency compression at the airway opening; and AD = autogenic drainage.

supplied with vests of appropriate sizes for tiny babies and has not been studied in this group.

When children reach about 3 years of age, additional techniques become applicable, patients can be taught how to exhale and to perform varied cough maneuvers and controlled breathing. The forced exhalation technique should be taught if dynamic compression is limiting clearance. Adding 'chicken breaths' (compressing the thorax by pressing the bent arms against the chest wall) to such a technique can make it more enjoyable for the child. Positive expiratory pressure mask application can be taught at this age, and rudimentary active cycle breathing can be applied. In Europe, 4- and 5-year-old children begin to learn autogenic drainage. In America, autogenic drainage has not been shown to be helpful until the pre-adolescent years. This may reflect as yet unstudied cultural differences between the North American and European groups of children.

Children near 12 years of age and older typically prefer independent techniques such as autogenic drainage and active cycle breathing. All techniques described in this paper are suitable alternatives for patients in these age groups. Exercise certainly can enter into the patient's life as soon as he or she begins to move about and should be added to the regimen of all patients. I believe that families who exercise together for fun develop strong habits to stay active over the years, benefiting everyone.

Patients with segmental atelectasis, particularly related to asthma, may benefit from PEP or PDPV. Prevention of postoperative atelectasis is also well suited to PEP; the major caveat here is that patients who have untreated or active pneumothorax should not perform PEP.

Neurologically abnormal patients who are unable to cooperate with any active method are also treated using PDPV and suctioning, if necessary. Musculoskeletal abnormalities—muscular dystrophies, myasthenia, poliomyelitis, or other similar diseases—require stabilization of bellows function. Optimizing ventilation in patients with such abnormalities may require positive pressure ventilation during sleep or continuously. Externally applied pressure, such as with the cyclically inflated pneumatic belt, can augment the patient's own efforts and is sometimes helpful. Normalizing the vital capacity and functional residual capacity typically helps to improve the ability to cough and clear secretions. Assisted cough devices or maneuvers are described in other papers from this conference by Bach[53] and Hill.[54] Not all patients who have weak muscles require nocturnal or continuous support and may benefit from PEP mask treatments. Further studies are sorely needed.

Bronchial hygiene in the home includes attention to exercise, general fitness, chest mobility, strength, and endurance, as well as added airway clearance techniques. Long-term controlled trials are urgently needed to help establish the best types of treatment for patients with cystic fibrosis and bronchiectasis. Such studies will become more complicated by the introduction of new treatments such as DNase and other therapies that alter secretions and may begin to change mucociliary clearance. Other airway secretion abnormalities also call for research; however, the selection of appropriate outcome measures is central to studying these questions, and it is unclear which are the most important. A recent meta-analysis of these techniques was presented at the 7th Annual North American Cystic Fibrosis Conference, in Dallas in 1993,[55] but few studies could be included due to differences in study design and lack of consistent adherence monitoring. We persist in prescribing therapy because patients over the years have found it helpful, but determining whether it is truly worthwhile (with randomized control studies that withhold treatment from control subjects) is now considered unethical. It seems intuitive that characteristics of mucus in different diseases affect clearance of secretions. The growing interest in new airway clearance techniques has had an additional benefit—stimulation of interest in mucus. Research to characterize mucus and to unravel the mysteries of how it travels through airways in normal and abnormal situations and how it can be changed is being addressed in laboratories across the country.

Clearer answers to many of these questions should be found in the coming decade.

REFERENCES

1. Frownfelter D. Postural drainage, percussion and vibration. In: Frownfelter D, ed. Chest physical therapy and pulmonary rehabilitation: an interdisciplinary approach, 2nd ed. Chicago: Year Book, 1987:271-288.
2. Kigin C. Chest physical therapy. In: Pierson DJ, Kacmarek RM, eds. Foundations of respiratory care. New York: Churchill Livingstone, 1992:777-792.
3. American Association for Respiratory Care. Clinical practice guideline: bronchial hygiene therapy. Respir Care 1991;36:1418-1426.
4. Schidlow DVS. The immotile cilia syndrome in children. Resident & Staff Physician 1983;29(6):34-40.
5. Holinger LD, Sanders AD. Chronic cough in infants and children: an update. Laryngoscope 1991;101(6, Part 1): 596-605.
6. Lewiston N. Bronchiectasis. In: Hilman B, ed. Pediatric respiratory disease. Philadelphia: WB Saunders Co, 1993:222-229.
7. Hardy KA. Advances in our understanding and care of patients with cystic fibrosis. Respir Care 1993;38(3): 282-289.
8. Le Mauviel L. Primary ciliary dyskinesia. West J Med 1991;155(3):280-283.
9. Lewiston N. Bronchiectasis in childhood. Pediatr Clin North Am 1984;31:865-878.
10. Hardy KW, Schidlow DV, Zaeri N. Obliterative bronchiolitis in children. Chest 1988;93:460-466.
11. Kamei RK. Chronic cough in children. Pediatr Clin North Am 1991;38(3):593-605.
12. Pryor JA, Webber BA, Hodson ME, Batten JC. Evaluation of the forced expiration technique as an adjunct to postural drainage in treatment of cystic fibrosis. Br Med J 1979;2(6187):417-418.
13. Murray JF. The ketchup bottle method (editorial). N Engl J Med 1979;300(20):1155-1157.
14. Eid N, Buchheit J, Neuling M, Phelps H. Chest physiotherapy in review. Respir Care 1991;36(4):270-282.
15. Zapetal A, Stefanova J, Horak J, Vavrova V, Samanek M. Chest physiotherapy and airway obstruction in patients with cystic fibrosis: a negative report. Eur J Respir Dis 1983;64(6):426-433.
16. Murphy MB, Concannon D, FitzGerald MX. Chest percussion: help or hindrance to postural drainage? Irish Med J 1983;76(4):189-190.
17. Selsby DS. Chest physiotherapy. Br Med J 1989;298 (6673):541-542.
18. Pryor JA, Webber BA, Hodson ME. Effect of chest physiotherapy on oxygen saturation in patients with cystic fibrosis. Thorax 1990;45(1):77.
19. Feldman J, Traver GA, Taussig LM. Maximal expiratory flows after postural drainage. Am Rev Respir Dis 1979; 119(2):239-245.
20. Desmond KJ, Schwenk WF, Thomas E, Beaudry PH, Coates AL. Immediate and long-term effects of chest physiotherapy in patients with cystic fibrosis. J Pediatr 1983;103(4):538-542.
21. Reisman JJ, Rivington-Law B, Corey M, Marcotte J, Wannamaker E, Harcourt D, Levison H. Role of conventional physiotherapy in cystic fibrosis. J Pediatr 1988; 113(4):632-636.
22. Pryor J. The forced expiratory technique. In: Pryor J, ed. Respiratory care. London: Churchill Livingstone, 1991: 79-100.
23. Miller S, Hall D, Clayton CB, Nelson R. Chest physiotherapy in cystic fibrosis: a comparative study of autogenic drainage and active cycle of breathing technique (formerly FET) (abstract). Pediatr Pulmonol Suppl 1993;9:267.
24. Mahlmeister MJ, Fink JB, Hoffman GL, Fifer LF. Positive-expiratory-pressure mask therapy: theroretical and practical considerations and a review of the literature. Respir Care 1991;36(11):1218-1229.
25. Andersen JB, Qvist J, Kann T. Recruiting collapsed lung through collateral channels with positive end-expiratory pressure. Scand J Respir Dis 1979;60(5):260-266.
26. Andersen JB, Falk M. Chest physiotherapy in the pediatric age group. Respir Care 1991;36(6):546-554.
27. Mortensen J, Falk M, Groth S, Jensen C. The effects of postural drainage and positive expiratory pressure physiotherapy on tracheobronchial clearance in cystic fibrosis. Chest 1991;100(5):1350-1357.
28. Oberwaldner B, Evans JC, Zach MS. Forced expirations against a variable resistance: a new chest physiotherapy method in cystic fibrosis. Pediatr Pulmonol 1986;2(6): 358-367.
29. Chevaillier J. Autogenic drainage. In: Lawson D, ed. Cystic fibrosis: horizons. Chichester MA: John Wiley, 1984:235.
30. Kraemer R, Zumbuhl C, Rudeberg A, Lentze MJ, Chevaillir J. 'Autogene drainage' bei Patienten mit zystischer Fibrose. Padiatr Prax 1986;33:223-232.
31. Spence S, Anderson B, Hardy K. Use of biofeedback to teach autogenic drainage (abstract). Pediatr Pulmonol Suppl 1990;5:A332.

32. Schoni MH. Autogenic drainage: a modern approach to physiotherapy in cystic fibrosis. J R Soc Med 1989;82 (Suppl 16):32-37.
33. McIlwaine M, Davidson AGF, Wong LTK, Pirie G. The effect of chest physiotherapy by postural drainage and autogenic drainage on oxygen saturation in cystic fibrosis (abstract). Pediatr Pulmonol Suppl 1991;6:291.
34. Warwick WJ, Hansen LG. The long-term effect of high-frequency chest compression therapy on pulmonary complications of cystic fibrosis. Pediatr Pulmonol 1991; 11(3):265-271.
35. Freitag L, Kim CS, Long WM, Venegas J, Wanner A. Mobilization of mucus by airway oscillations. Acta Anaesthesiol Scand Suppl 1989;90:93-101.
36. King M, Phillips DM, Zidulka A, Chang HK. Tracheal mucus clearance in high-frequency oscillation. II. Chest wall versus mouth oscillation. Am Rev Respir Dis 1984; 130(5):703-706.
37. Rubin EM, Scantlen GE, Chapman GA, Eldridge M, Menendez R, Wanner A. Effect of chest wall oscillation on mucus clearance: comparison of two vibrators. Pediatr Pulmonol 1989;6(2):122-126.
38. Ahrens R, Gozal D, Omlin KJ, Vega J, Boyd KP, Woo MS, Keens TG. Comparative efficacy of high frequency chest compression and conventional chest physiotherapy in hopitalized patients with cystic fibrosis (abstract). Pediatr Pulmonol Suppl 1993;9:267.
39. Hüls G, Lindemann H. Vergleichende Untersuchungen zur Physiotherapie: flutter VRP 1 versus autogene drainage [comparison between two physiotherapeutic methods: the flutter VRP 1 and autogenic drainage]. Atemw und Lungenkrankheiten 1991;17:414.
40. Lindemann H. Zum Stellenwert der Physiotherapie mit dem VRP 1-Desitin ("Flutter") [The value of physical therapy with VRP 1 ("Flutter")]. Pneumologie 1992; 46(12):626-630.
41. Zach MS, Purrer B, Oberwaldner B. Effect of swimming on forced expiration and sputum clearance in cystic fibrosis. Lancet 1981;2(8257):1201-1203.
42. Zach M, Oberwaldner B, Hausler F. Cystic fibrosis: physical exercise versus chest physiotherapy. Arch Dis Child 1982;57(8):587-589.
43. Smaldone GC, Itoh H, Swift DL, Wagner HN Jr. Effect of flow-limiting segments and cough on particle deposition and mucociliary clearance in the lung. Am Rev Respir Dis 1979;120(4):747-758.
44. Oldenburg FA Jr, Dolovich MB, Montgomery JM, Newhouse MT. Effects of postural drainage, exercise, and cough on mucus clearance in chronic bronchitis. Am Rev Respir Dis 1979;120(4):739-745.
45. de Boeck C, Zinman R. Cough versus chest physiotherapy: a comparison of the acute effects on pulmonary function in patients with cystic fibrosis. Am Rev Respir Dis 1984;129(1):182-184.
46. Bain J, Bishop J, Olinsky A. Evaluation of directed coughing in cystic fibrosis. Br J Dis Chest 1988;82(2): 138-148.
47. Passero MA, Remor B, Salomon J. Patient-reported compliance with cystic fibrosis therapy. Clin Pediatr Phila 1981;20(4):264-268.
48. Maxwell M, Redmond A. Comparative trial of manual and mechanical percussion technique with gravity-assisted bronchial drainage in patients with cystic fibrosis. Arch Dis Child 1979;54(7):542-544.
49. Falk M, Mortensen J, Kelstrup M, Lanng S, Larsen L, Ulrik CS. Short-term effects of positive expiratory pressure and the forced expiration technique on mucus clearance and lung function in CF (abstract). Pediatr Pulmonol Suppl 1993;9:268.
50. Whitman J, Van Beusekom R, Olson S, Worm M, Indihar F. Preliminary evaluation of high-frequency chest compression for secretion clearance in mechanically ventilated patients. Respir Care 1993;38(9):1081-1087.
51. Bolek JE. Variations of positive expiratory pressure (abstract). Pediatr Pulmonol Suppl 1993;9:93-94.
52. Henke KG, Orenstein DM. Oxygen saturation during exercise in cystic fibrosis. Am Rev Respir Dis 1984; 129(5):708-711.
53. Bach JR, Saporito LR. Indications and criteria for decannulation and transition from invasive to noninvasive long-term ventilatory support. Respir Care 1994;39(5): 515-531.
54. Hill NS. Use of negative pressure ventilation, rocking beds, and pneumobelts. Respir Care 1994;39(5):532-549.
55. Thomas J. The efficacy of physical therapy modalities in the treatment of patients with CF: a meta-analysis. In: Proceedings of the 7th Annual North American Cystic Fibrosis Conference. Oct 13-16, 1993, Dallas TX.

Hardy Discussion

Bach: Karen, why not offer these different methods to the COPD or cystic fibrosis patient, and then ask the patient which works best? Sure, you can get up a lot of mucus, but some of these methods may be causing secretions, so why don't we find out from the patient what works best and give him the choice? The other thing I wanted to mention was the Percussionator (Percussionaire Corp, Sandpoint ID).

Stoller: Percussionator?

Bach: It gives high pressure pulses at 2-7 Hz, with a high I-E ratio, so the patient can't fully exhale. It's meant to be percussing the lung. I think the FDA passed it recently for use in the U.S.

Hardy: You use this at the mouth or on the body?

Bach: At the mouth.

Stoller: Does this technique ever result in bronchospasm?

Hardy: An increase in secretion production is certainly something that you have to watch out for in these patients. Any of these techniques by movement of mucus can theoretically provoke an irritative response with bronchospasm in any of our patients that we're discussing. Just as postural drainage, percussion, and vibration (PDPV) has been shown to adversely affect some patients, newer airway clearance techniques (ACTs) need to be closely evaluated for adverse effects.

Hill: I think your approach to the patient would differ depending on what the basic problem is, to a certain extent. I think a lot of what you've talked about really relates to the cystic fibrosis population; if you consider a neuromuscular disease population that has normal mucus, a normal mucociliary escalator, and a very weak cough, things might be quite different. I think John Bach probably has more experience in this than anybody else, but, in my experience, techniques that assist expiratory muscle effort are of more benefit than anything else, such as manually assisted coughing or the insufflator/exsufflator. The BiPAP™ device, on the other hand, wouldn't have been on my list of things that would help patients with cystic fibrosis raise secretions.

Hardy: BiPAP™ can help to preserve normal volumes. In those patients who can't even take a good breath in, anything else you do that ends up helping them achieve normal ventilation can also help them generate a more effective cough. The insufflator/exsufflator or manual assistance and even 'chicken breaths' can help kids with these problems. There's a long, long list of what can be used in that particular population that I'm sure John will review in great detail.

Hill: One other question—how do mucolytics, DNase, and so forth, fit into this?

Hardy: DNase has been wonderful in one way—everybody's excited about mucus and studying mucus per se because of mucolytics. With the resurgence of some of these therapies, like the idea of DNase or rehydrating secretions by altering ion flux in the respiratory epithelium with things like amiloride and UTP, investigators are receiving funds to study clearance issues. All of us are thinking more about what is lying on top of cilia, and whether changing it is going to make things better or worse. Cystic fibrosis patients have increased concentrations of neutrophil DNA in their sputum. The theoretical function of DNase is to lyse the DNA, leaving the mucin lattice still intact, so that the cilia can move it a bit more easily. It does liquefy secretions, and in healthier patients that's a good thing. We clearly don't know enough yet about how DNase is going to work in the sicker or younger cystic fibrosis population. Many questions need to be applied to the studies that have been done so far. DNase is obviously going to be marketed very shortly, and will cost a fortune. The estimates now are $17,000 to $50,000 per patient just for a single dose (that is once a day dosing at 2.5 mg for a year). A clear look at the studies that have been published[1-3] and the hospital days saved (1.4 hospital days saved/6 mo, for the moderately severe patient with cystic fibrosis) do not show a favorable cost-benefit ratio. We have a lot more to learn about mucolytics and how mucolytic pharmacotherapy might affect airway clearance. DNase will be used for more than CF. Research on chronic bronchitis is soon to be published (personal communication, Vivika Hansen, Genentech, 1993). We don't know exactly how clearance methods will work in concert with this drug for each disease.

REFERENCES

1. Aitken ML, Burke W, McDonald G, Shak S, Montgomery AB, Smith A. Recombinant human DNase inhalation in normal subjects and patients with cystic fibrosis: a phase-I study. JAMA 1992;267(14):1947-1951.
2. Hubbard RC, McElvaney NG, Birrer P, Shak S, Robinson WW, Jolley C, et al. A preliminary study of aerosolized recombinant human deoxyribonuclease I in the treatment of cystic fibrosis. N Engl J Med 1992;326(12):812-815.
3. Ramsey B, for the Pulmozyme (rhDNase) Study Group. A summary of the results of the Phase III multicenter clinical trial: aerosol administration of recombinant human DNase reduces the risk of respiratory tract infections and improves pulmonary function in patients with cystic fibrosis (abstract). Presented at the 7th

Annual North American Cystic Fibrosis Conference held in Dallas, Texas, October 15, 1993. Pediatr Pulmonol Suppl 1993;9:152-153.

Make: I have two questions. First, what are the effects on secretions of bronchodilators or saline by nebulizer versus metered-dose inhaler? Second, I've been impressed recently because I've used PEP therapy on a lot of patients with obstructive lung disease, including bronchiectasis, and it seems to be beneficial. What is the mechanism of PEP therapy? None of the mechanisms you suggested make sense to me, unless I'm missing something.

Hardy: I would say most ACT studies[1-3] do not control for use, timing, or type of bronchodilator therapy at time of pulmonary function testing. Obviously, bronchodilators can change pulmonary function, and many patients with cystic fibrosis, for example, have hyperreactive airways responsive to bronchodilators. The specific question of whether a nebulized treatment might help an ACT work better than an MDI hasn't been studied. Your second question was how does PEP really work? A few theories have been put forth.[4] I think the most understandable thing about PEP is the airway pressure change. The device generates a positive expiratory pressure that moves the equal pressure point during exhalation. This can do two things. First, while the patient has positive pressure exhalation, more air can move through collateral channels to underaerated alveoli. Second, the change in equal pressure point (EPP) can allow clearance of secretions by air-liquid interactions in areas that were previously collapsed.

REFERENCES

1. Desmond KJ, Schwenk WF, Thomas E, Beaudry PH, Coates AL. Immediate and long-term effects of chest physiotherapy in patients with cystic fibrosis. J Pediatr 1983;103(4):538-542.
2. Eid N, Buchheit J, Neuling M, Phelps H. Chest physiotherapy in review. Respir Care 1991;36(4):270-282.
3. Oberwaldner B, Evans JC, Zach MS. Forced expirations against a variable resistance: a new chest physiotherapy method in cystic fibrosis. Pediatr Pulmonol 1986;2(6):358-367.
4. Mahlmeister MJ, Fink JB, Hoffman GL, Fifer LF. Positive-expiratory-pressure mask therapy: theoretical and practical considerations and a review of the literature. Respir Care 1991; 36(11):1218-1229.

Make: You are using the positive expiratory pressure only during inhalation of a bronchodilator. But then you discontinue positive pressure and cough afterwards?

Hardy: During the time that you're using the valve, the equal pressure point is going to be changed, so that you're going to keep airways distended. Theoretically, if the patient has any remaining mucociliary escalator, mucus that previously was stuck can move. While you're doing the PEP part, you start increasing the air in underaerated alveoli in the distal portions of the lung, through collateral ventilation. This supplies air power behind that mucus plug, so that when you do cough, you'll be pushing that mucus forward. Jens Andersen,[1] in Denmark, took cadaver lungs and applied pressure to re-expand atelectatic lungs. He made molds and demonstrated that pressures ≥ 15 cm H_2O were able to re-expand alveoli through the pores of Kohn. Theoretically, when these patients subsequently cough, avoiding dynamic compression with a huff maneuver, alveoli are squeezed upon and air pushes through airways that previously were obstructed. Air moves mucus forward a bit.

REFERENCE

1. Andersen JB, Jesperson W. Demonstration of intersegmental respiratory bronchioles in normal human lungs. Eur J Respir Dis 1980;61(6):337-341.

Make: It seems to me that you may also be aerating relatively nonaerated portions of lungs by increasing the expiratory pressure.

Celli: I love these controversies, because on the one hand we have John Bach in every discussion saying we should ask the patient, and on the other hand we have mortality as an end point. The reason why I bring it up is that in the only study that I know of where they looked at how patients felt, the Organidin (iodinated glycerol) study of Tom Petty's, the patients said, candidly, "I feel better, things are better." So, I'd like to have your opinion on whether this has been seen in cystic fibrosis.

REFERENCE

1. Petty T, chairman. The National Mucolytic Study: results of a randomized, double-blind, placebo-controlled study of iodinated glycerol in chronic obstructive bronchitis. Chest 1990; 97(1):75-83.

Hardy: Yes, that same questionnaire was utilized in a comparative study with postural drainage, percussion, and vibration; autogenic drainage; and PEP for cystic fibrosis patients by McIlwaine and Davidson's group from Vancouver.[1] Patients clearly preferred the independent methods over conventional therapy, and they liked autogenic drainage. Autogenic drainage cleared the most sputum, closely followed by PEP, and then, least effective, was conventional therapy [PDPV]. When asked what they preferred, they clearly preferred an independent technique—these were young adults—to a dependent technique, and they felt better when they were doing these independent techniques, for whatever that's worth. I agree with you, John (Bach). I think that variety is the spice of life. If they're all equivalent, which is sort of the way the studies are coming out now, if they're all pretty equivalent to our current gold standard, then why not let the patient do what they like, that they can do well, and that seems to work for them. It's also the case that as your disease changes, even in one time of exacerbation, maybe when you come into the hospital, you can't do autogenic because you're too sick. But after a few days you start feeling better, you've gotten conventional therapy for a few days, you're ready to go to an independent technique again. So that, even in a short period of time, you might change what you think is the right treatment for you.

REFERENCE

1. McIlwaine M, Davidson AGF, Wong LTK, Pirie G. The effect of chest physiotherapy by postural drainage and autogenic drainage on oxygen saturation in cystic fibrosis (abstract). Pediatr Pulmonol Suppl 1991;6:291.

Celli: Have you tried Organidin? Mucokinetics?

Hardy: No.

Make: What are the indications for initiating these different techniques?

Hardy: I can speak for cystic fibrosis and then in general. I think that one first decides whether a disease process is present and whether its presence is a reason not to clear secretions normally. Do they have something wrong with their mucociliary escalator? Are they in a hypersecretory state? Can they cough? Are they in a situation where theoretically they're going to have abnormal clearance? And once you get past this, timing of treatment is the next hurdle—even taking patients, for example, with cystic fibrosis, we hotly debate timing all the time. Infants with CF are born with anatomically normal lungs, but in their first 6 months already show mucus obstruction of airways. They do start obstructing quickly, over a short period of time. Bronchoscopic studies in youngsters show uninfected lungs but lots of mucus plugging.[1] PFT data on young infants also documents early obstruction.[2,3] This fuels the argument for treating the asymptomatic child. If you're going to be very aggressive, you want to stop the cycle before it begins. We start an airway clearance technique very early in life, and very early in life we don't have too much choice. We're usually stuck with postural drainage, percussion, and vibration.

REFERENCES

1. Wood R. Treatment of CF lung disease in the first 2 years (abstract). Pediatr Pulmonol Suppl 1989;4:68S-70S.
2. Tepper RS, Hiatt P, Eigen H, Scott P, Grosfeld J, Cohen M. Infants with cystic fibrosis: pulmonary function at diagnosis. Pediatr Pulmonol 1988; 5(1):15-18.
3. Tepper RS, Hiatt P, Eigen H, Smith J. Total respiratory system compliance in asymptomatic infants with cystic fibrosis. Am Rev Respir Dis 1987; 135(5):1075-1079.

Make: I'm worried about how this message may come across to our readers because you haven't really talked about indications, and cystic fibrosis is different from other diseases. Take a patient with anatomic bronchiectasis that you demonstrate noninvasively by CT scan, but who doesn't have any cough or sputum production—that patient to me doesn't necessarily have any indication to begin to think about these techniques.

Hardy: That brings up Murray's 1979 ketchup bottle theory and his statement as well as many others who say that if you're not producing at least 30 mL sputum a day, you shouldn't be doing an airway clearance technique because it's not worth it. If you look at these new studies of the independent modes of drainage, like PEP or autogenic drainage, you can find many patients who have bronchiectasis (in this case with cystic fibrosis) who don't produce any sputum with conventional therapy. They produce more than 30 mL easily with one of these techniques. You are just fooling yourself that those patients don't really need treatment because there's no ketchup in the bottle. Yes, there is ketchup in the bottle, you know there's ketchup in the bottle, and you need to do something to go get it. If they have a recurrent cycle of infection and disease and you know they have bronchiectasis, I think that you can make a rational physiologic argument for why that patient should have an airway clearance technique—now! You can always try one of these newer methods that is prone to produce extra sputum and find out whether you produce sputum in your patient. If all of a sudden they're bringing up a lot of sputum, you should continue.

Leger: Do you think there is a place for IPPB in improving secretion clearance, and if so in what patient population? My idea is that by improving inspiratory volume and increasing expiratory flow, cough effectiveness and therefore secretion clearance will be improved. What are your thoughts on that?

Hardy: You can probably all address that question better than I because that's something we rarely use in pediatrics. But on the theory that you may be able to improve aeration in underaerated units with IPPB and subsequently be able to cough and clear secretions better—under that argument I can see a place for IPPB, strictly in John's (Bach) patients. I'm sure you'll talk about that. I have limited experience with it.

Transition from the Intensive Care Unit to Home: Patient Selection and Discharge Planning

Mary Gilmartin BSN RRT

- **I. Introduction**
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- **VI. Discharge Planning**
  - **A. Team Concept**
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- **VIII. Home Care Agencies & Community Services**
- **IX. In Conclusion**

Introduction

Patients in the intensive care setting who remain dependent on mechanical ventilatory support pose a unique dilemma to the physicians and allied health professionals responsible for their care and to government agencies and the private health insurance industry that are reimbursing for this expensive care. Should these patients remain in the intensive care setting? If they can make the a transition from the ICU, where will they be placed? Who will make the decision? Will it be the physician, the insurance company, the family, the discharge planner, or a utilization review committee? If the patient is a candidate for home care, in what setting will discharge planning take place? Will the patient be rehabilitated prior to discharge? Are there specific physiologic or psychosocial indicators that predict successful outcomes for long-term ventilator patients? Can the family take on the responsibility of caring for the

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patient? Are there adequate resources for the patient and family? How will a safe environment be assured? What are the psychological implications of long-term ventilator care?

The healthcare team, the patient, and the family are faced with these issues and many others when there is the need for long-term ventilatory support and/or the inability to be weaned from ventilatory support.

Outcomes for Ventilator-Dependent Patients

The successful outcomes of patients discharged to home on ventilatory support provide us with data that help us determine patients who are appropriate for home care, but information in the recent literature regarding outcomes is limited. Data collected on nonoperative patients who were ventilated for at least 7 days showed a hospital mortality rate of 57%.[1] These patients had mean APACHE II scores of 11-15.[2,3] (The APACHE scoring system correlates acute physiology, age, and chronic health data to determine the appropriateness of critical care and the likelihood of patient survival. Each factor is weighted according to its effect on survival. The higher the number the heavier the weight; the higher the total score the less the likelihood of survival.) Other patients who were ventilated for a comparable length of time but who had higher APACHE II or lower APACHE II scores had significantly increased (77%) or decreased (10%) mortality rates. Data from the 12 institutions participating in the study[1] reported by Knaus reflect that in mechanically ventilated patients higher mortality is associated with increased severity of illness. The underlying disease process causing the need for mechanical ventilation contributes greatly to the outcome. Robert et al[4] have shown that the survival of patients with obstructive lung disease (COPD) receiving mechanical ventilation in the home after 4 years is 50% less than that of patients with restrictive disorders (Fig. 1). A study by my group,[5,6] reported in abstract form, suggests that the survival of COPD patients at home on ventilatory support was markedly reduced compared to survival of patients with neuromuscular disease (Fig. 2). We also showed that patients with COPD had more frequent and longer hospital readmissions.

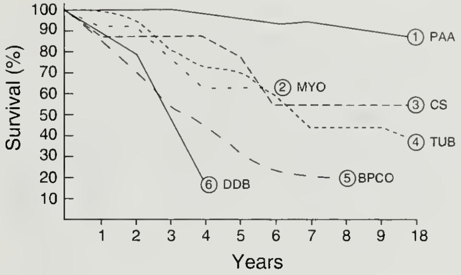

Fig. 1. Actuarial survival in years of patients receiving home mechanical ventilation. 1 PAA = 41 postpolio patients; 2 MYO = 13 patients with myopathies; 3 CS = 53 patients with kyphoscoliosis; 4 TUB = 55 patients with sequelae of tuberculosis; 5 BPCO = 50 patients with chronic obstructive lung disease; 6 DDB = 10 patients with bronchiectasis. (From Reference 4, with permission.)

The Health Care Financing Administration's (HCFA) chronic ventilator-dependent demonstration study is being conducted at four sites in the United States and focuses on the management of ventilator patients in a nonintensive care setting.[7] Experience from these demonstration sites will enhance our understanding of the impact of non-ICU care by a multidisciplinary team, determine which patients are ventilator dependent, and provide long-term data on survival after discharge. Preliminary data from two of these units (Temple University

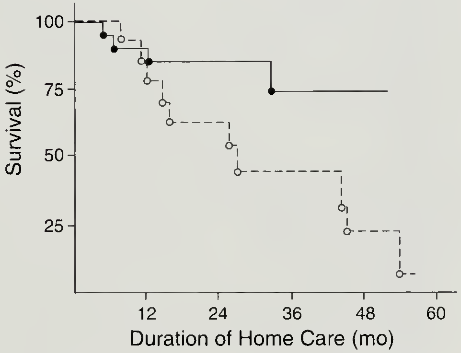

Fig. 2. Survival of adults receiving mechanical ventilation in the home from 1981-1987. NM = 21 patients with neuromuscular disease; COPD = 15 patients with chronic obstructive pulmonary disease. (From Reference 6, with permission.)

Medical Center[8] and the Mayo Clinic[9]) are shown in Table 1. The preliminary report from Criner et al[8] does not specify which patients needed continuous ventilatory support or what is meant by functional status improvement. One can speculate (but it is only speculation) that the patients who required continuous ventilation had underlying neuromuscular disease or advanced lung disease.

Long-term results from the Mayo Unit[9] have not been analyzed because the unit has been open for only a short time. Both Criner et al[8] and Gracey et al[9] report an improvement in nutritional status from admission to discharge as measured by serum albumin levels with the hypoalbuminemia on admission attributed to catabolic status and decreased protein synthesis associated with severe illness.

Make and I[10] have published information, gathered through the National Center for Home Ventilation, on 232 individuals using positive pressure ventilation at home (Fig. 3). This report documents a mean length of time in the home of 49.8 months with 14 patients at home for more than 10 years. Most of the patients have neuromuscular diseases (64% adults, 56% children), and 17% have COPD.

A survey of patients requiring long-term ventilatory support was conducted in Minnesota in 1986 and again in 1992[11] to determine the prevalence and characteristics of patients at home and in long-term care facilities. In 1986, 103 individuals were identified, with 83 of them cared for in the home; and in 1992, 216 were identified with 143 at home (an increase of 110%). Of the total, patients cared for in long-term care facilities increased from 19% to 35%. In 1986, only 1 long-term care facility existed in Minnesota, but in 1992 the number had grown to 4 and the number of home medical equipment (HME) companies caring for long-term ventilator patients had increased from 5 to 12. The largest numbers of patients had ventilatory muscle disorders (such as poliomyelitis, cervical trauma, and amyotrophic lateral sclerosis, or ALS). Of the patients with 'polio,' 9 have required ventilatory support for more than 20 years whereas the average length of time for patients with COPD was 1.9 years. The authors hypothesized that the increase in long-term ventilatory support has resulted from an increase in the prevalence of diseases that result in respiratory insufficiency, increased utilization of

Table 1. Patient Data and Outcomes from Two Ventilator-Dependent Patient Demonstration Units*

Factor	Temple University Medical Center	Mayo Clinic
Number of patients (women)	58 (31)	61 (–)
Age [y, mean (SD), range]	62 (18), –	68 (14), 24-89
Reason for mechanical ventilation		
Postoperative failure	15 (26%)	40 (67%)
Medical failure	43 (74%)	21 (33%)
Condition giving rise to failure		
Neuromuscular disease	15 (26%)	10 (16%)
Primary lung disease	17 (29%)	14 (23%) 8 restrictive, 6 COPD
Heart disease	3 (5%)	Unreported
Other	23 (40%)	37 (61%)
Number surviving to unit discharge	54 (93%)	58
Pre-unit ventilator days, range	33 (25), –	34 (24), 7-150
Unit ventilator days, range	49 (45), –	16 (12), 2-56
Discharge destination, n (%)*		
Home	55	35
Rehabilitation unit	31	8
Chronic care facility	7	15
Ventilator status at unit discharge		
Ventilator independent	46	48
Ventilator dependent	7 (12%)	0
Nocturnally ventilated	3 (5%)	5

*Information in this table is from References 8 and 9, except for the discharge destination data from Temple Medical Center, which comes from personal communication between GJ Criner MD and the author (1993).

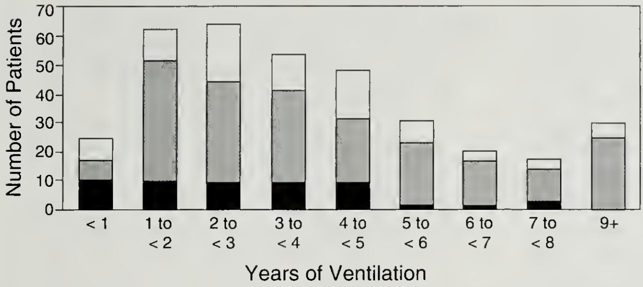

Fig. 3. Duration of home mechanical ventilation in 232 subjects. Mean duration was 49.8 months, with 14 patients at home for > 10 years. Average daily ventilator use was 16.5 hours and one half of patients required supplemental oxygen. ■ = COPD; ▨ = neuromuscular; and ☐ = other.

this supportive therapy by physicians; and better coordination, availability, and reimbursement for services.

In 1990, the American Association for Respiratory Care commissioned the Gallup organization to conduct telephone interviews with 300 hospital respiratory care department managers and 100 pulmonary physicians to determine the numbers of chronic ventilator patients, the costs incurred by these patients, and the discharge settings.[12] A chronic ventilator patient was defined as one needing ventilation for at least 6 hours/day for 30 or more days. It can be estimated from this study that in 1990 there were approximately 11,419 chronic ventilator patients in our hospitals. The daily cost of caring for these patients was $9,012,322 and hospitals absorbed about 46% of the cost of care. The survey found that once it had been determined that a patient could be sent to a post-acute care facility, a waiting period of approximately 35 days followed before a bed became available at an additional cost of $27,615.

I reported on 85 ventilator-assisted patients admitted to the respiratory care center at Boston University Medical Center from 1981-1988,[13] and the data are summarized in Table 2.

Fischer and Prentice[14] in a 1982 paper reported an 88% reduction in hospital days in patients with restrictive disorders and a 37% reduction in patients with COPD, after initiation of home ventilation. A summary of other experiences with home ventilation is given in Table 3.[15-19]

Table 2. Information on 85 Ventilator-Assisted Patients* followed at Boston University Medical Center from 1981-1988

	Reason for Mechanical Ventilation		
	COPD n = 37	Neuromuscular Disease n = 39	Other n = 9
Age (years)			
Mean	60.9	44.1	65.0
Range	46-85	22-84	39-79
Outcome			
Died in hospital	8	1	–
Discharged to chronic care facility	6	4	1
Discharged to home	23	34	8
Survivors at home (1988)	10	25	8
Survival duration (months)			
Range 1-72	3-82	1-24	
Mean 26.8	37.4	10.1	
Deaths after discharge to home	13	9	–
Survival time before death			
Mean (months)	25.2	18.8	

*Average daily ventilator use = 16.5 hours; one half of patients used supplemental oxygen.

Table 3. Duration of Mechanical Ventilation in the Home

First Author	n	Condition	Ventilator Type	Length (years)
Splaingard[14]	26	Neuromuscular disease	Positive pressure	Range 1-11
Splaingard[15]	40	Neuromuscular disease	Negative pressure	Range 0.5-19
Garay[16]	6	Alveolar hypoventilation syndromes	Negative pressure	Mean > 10
Bach[17]	257	Post-poliomyelitis	Noninvasive positive pressure (mouthpiece or mask)	Mean 13.5
Baydur[18]	3/17	Duchenne's dystrophy	Negative & positive pressure	Mean > 10

Patient Selection for Long-Term Ventilator Care

Primary Diagnosis

From this brief review of some of the published outcomes of long-term care, it is clear that patients with a primary diagnosis of neuromuscular and/or skeletal disease are the better candidates for long-term support. Candidates for long-term mechanical ventilation may be (1) patients who can make no spontaneous breathing effort or whose efforts are severely impaired, (2) patients who have failed repeated attempts at weaning after an acute episode of respiratory failure, and (3) patients who have a disorder causing chronic respiratory failure that requires repeated admissions or that severely limits their functional status. The patients in the first group may have central alveolar hypoventilation, spinal cord injury, intracranial hemorrhage, or stroke. These patients cannot survive without ventilatory assistance. The patients in the second category may have had a major insult to the respiratory system (as a result of severe illness or from postoperative complications) or may have an acute illness superimposed on a chronic respiratory disorder. Patients in the third group may have kyphoscoliosis, COPD, or progressive neuromuscular disorders. It is my impression that these patients may do best if ventilated electively prior to repeated episodes of respiratory failure because each admission to the ICU place the patient at risk for complications that may increase the length of stay and the medical costs for the admission. Many of these patients are also candidates for noninvasive ventilation, with the possibility of fewer complications, easier initiation, and improved quality of life.

Table 4[20] lists diseases by category for which long-term ventilation may be needed. Patients with neuromuscular disease may remain relatively stable over a long period of time with their underlying disease progressing only slowly. They usually do not have airflow limitations, their ventilatory status may deteriorate slowly, and most do not require chronic oxygen therapy. More recently, these patients have begun to choose elective ventilation via noninvasive positive pressure techniques.[21] Because not all neuromuscular diseases progress slowly, the decision to ventilate must be individualized and discussed at length with each patient. ALS is one disease that not only can be rapidly progressive but also may lead to loss of swallowing and speech early in the course of the disease. Many patients become depressed and frustrated over the inability to communicate basic needs. Healthcare professionals have a responsibility to communicate honestly with all patients who are moving toward the need for ventilatory support.[21] The patient and family can make an informed decision only when they know all the facts related to the illness, ventilation, and resuscitation. The patient does need to be ready to hear the information, and repeated discussions will be necessary. We have an obligation to discuss living wills and other advance directives with patients who have none and to encourage periodic review of existing advance directives by those who do have them. Silverstein et al[21] believe that patients may change their minds about the use of life-sustaining support because of experience with the illness, changes in subjective evaluation of quality of life, and changes in their evaluation of the benefits and burdens of life-sustaining technology.

Table 4. Conditions that May Necessitate Long-Term Mechanical Ventilation*

Neuromuscular Disorders
- Central nervous system
  - Central hypoventilation syndromes
  - Ondine's Curse
  - Arnold-Chiari malformation

- Spinal cord
  - Traumatic injuries
  - Thoracic myelomeningocele
  - Syringomyelia

- Anterior Horn Cell (lower motor neuron)
  - Poliomyelitis
  - Spinal muscle atrophy (Werdnig-Hoffman)
  - Amyotrophic lateral sclerosis†

- Muscle
  - Muscular dystrophy (Duchenne's, limb girdle, myotonic dystrophy)
  - Congenital myopathies

- Peripheral nerve
  - Phrenic neuropathies
  - Diaphragmatic paralysis
    - Idiopathic
    - Postsurgical
  - Guillain-Barré syndrome

Chest-Wall and Diaphragmatic Defects
- Kyphoscoliosis
- Postsurgical (thoracoplasty)
- Diaphragmatic hernia

Primary Pulmonary Disorders
- Tracheomalacia
- Bronchiectasis
- Bronchopulmonary dysplasia
- Chronic aspiration
- Chronic bronchitis, emphysema*
- Cystic fibrosis*
- Interstitial lung disease (multiple causes)*
- Adult respiratory distress syndrome

*From Reference 20, with permission.
†Less appropriate disorders for home mechanical ventilation.

One major category of patients that raises much controversy are those with primary pulmonary disease. Most patients with end-stage COPD are older, have pulmonary hypertension, cor pulmonale, severe unrelenting dyspnea, and rapid fluctuations in airflow obstruction with resultant hypoxemia and hypercarbia. Even when these patients are mechanically ventilated, it may not be possible to achieve stable levels of ventilation and oxygenation. The decision to ventilate patients with COPD must be made carefully, and the patient must be fully aware of the potential benefits and disadvantages. The clinician may want to consider a trial of noninvasive nasal mask ventilation to monitor the clinical response and patient tolerance of the procedure. The patient who cannot tolerate a mask secured by headstrap most likely will not do well with a tracheostomy. Patients with cystic fibrosis are not considered good candidates because of the problems with pulmonary secretions and infection (although noninvasive positive pressure ventilation may be initiated, especially if the patient is awaiting lung transplantation). Patients with pulmonary fibrosis may still experience dyspnea and an increased work of breathing even when high oxygen concentrations and high inflating pressures maintain minimally acceptable blood gas values.[22]

Comorbidity

The primary reason for a patient's becoming ventilator dependent only partially answers the question of whether that patient is a candidate for home care. Other medical illnesses and chronic conditions may also play a role in how well the patient will do over time in the home environment. A patient with a spinal cord injury but without other medical problems may do well with long-term ventilation provided he does not develop related problems, such as urosepsis, renal calculi, or major decubiti. Older patients are likely to have more coexisting medical problems.

Cardiac disease coupled with chronic respiratory failure and the need for ventilatory support may place the patient at high risk for medical emergencies in the home and the need for significant medical interventions. Patients in this category are not good candidates for home care unless their cardiac disease is responsive to medical management and stable prior to discharge. A cardiac workup may reveal severe ventricular dysfunction and/or arrhythmias that would impair the patient's ability to become more independent in functional activities.

Patients with metastatic cancer or other terminal conditions cannot be considered for home care unless full-time professional support is available in the home; otherwise, by the time the family is able to provide the necessary care the patient may be too close to death to go home. The expense of equipment and supplies to be used in the home may outweigh the wish of patient and family to have the patient spend his last days at home. The insurance carrier may not consider home care to be cost-beneficial in these circumstances.

Other chronic illnesses such as renal failure or liver disease may significantly impede the patient's ability to regain sufficient strength or stability to return home. Anemia decreases oxygen carrying capacity and if severe may limit the patient's endurance. Patients with marked psychiatric illness who are not stabilized with medications and/or psychiatric therapy are not good candidates for home care unless the family is willing to provide full-time care. If patients are unable to make good judgments about self-care and equipment, they are at risk for major problems in the home.[20] Each situation must be individualized and the decision based on all aspects of the patient's physical, psychological, and family support systems.

Criteria for Discharge to the Home

Clinical & Physiologic Stability

In order to optimize patient safety in the home, the patient should be clinically and physiologically stable for at least 2-4 weeks prior to discharge[22] (Table 5[20]). The patient should not require changes in ventilator settings unless those changes are needed only during periods of increased activity or to improve phonation with tracheostomy-cuff deflation. Some patients may need to increase oxygenation during exercise or activities of daily living or to increase the tidal volume when the cuff is deflated to allow normal speech. These particular ventilator changes must be initiated when the patient is hospitalized and appropriately monitored. Changes should not be needed for episodes of severe dyspnea or tachypnea, and the patient's underlying dyspnea related to the primary disease must be under control. Arterial blood gas values must be acceptable at the patient's current ventilator settings. When a patient requires an inspired oxygen concentration (F_{IO_2}) of greater than 0.40, providing home care becomes more complex and expensive, and the need may reflect an underlying clinical condition that increases the respiratory care needs of the patient. When positive end-expiratory pressure (PEEP) is needed to maintain satisfactory gas exchange or treat segmental or lobar atelectasis, the complexity of the ventilator system and the educational needs of the patient and the caregivers are increased. PEEP valves are easily dislodged from the exhalation valve, they are not always accurate, and the setting may be changed inadvertently. The patient's work of breathing may also be increased if the ventilator sensitivity is not properly adjusted. PEEP levels of < 10 cm H_2O [0.981 kPa] may be necessary in the home, but the patient's clinical condition should be reassessed periodically to justify continued use. The patient should be on an assist-control or pressure-limited (for pediatric patients) mode of ventilation rather than intermittent mandatory ventilation (IMV). Prior research has shown that the IMV mode on portable ventilators increases the work of breathing, and when modifications are added to provide sufficient flow for spontaneous breathing, the complexity of the equipment is increased.[23] Critical care ventilators are not appropriate for home care because of their complexity and the need for high pressure gas sources that greatly increase the operating cost.

Table 5. Patient Stability and Medical Readiness for Discharge*

Control or absence of sustained dyspnea

Acceptable arterial blood gases, with $F_{IO_2} < 0.40$

Stable ventilator settings

- $F_{IO_2} < 0.40$
- Assist/control or pressure-limited mode (pediatrics)
- Limited use of PEEP
- Minimal fluctuations in airway resistance and compliance
- Stable 'free time' periods

Optimal metabolic and acid-base status

Absence of acute infectious processes

Absence of life-threatening cardiac dysfunction or arrhythmias

Other organ systems stable

Ability to clear secretions and protect airway

Adequate nutrition

Progression of growth and development (in children)

If artificial airway, a tracheostomy, not an oral or nasal airway

Able to handle the daily stressors

Management at home expected to be stable, without the need for readmission within at least 1 month

*From Reference 20, with permission.

Airway resistance and compliance should change only minimally, with peak inspiratory pressures (PIP) not varying more than ± 5 cm H_2O. Changes in PIP should respond to suctioning, bronchodilator administration, removing water from the ventilator circuit, and minimizing tracheal cuff leaks.

If the patient is able to be off the ventilator for any length of time, these weaning periods must be stable. The patient should feel comfortable with the free time; it should be a routine part of the daily activities; and minimal changes in arterial blood gas values should be seen at the end of the weaning period. The mode of weaning should be simple for the patient and caregivers to manage. If the patient has been weaned in the hospital on a pressure support system, he needs to be switched to a T-piece or tracheostomy mask in advance of the anticipated discharge date.

Patients who have had prolonged ventilatory support may have associated medical problems that must be stabilized prior to discharge. Major diagnostic and therapeutic interventions should not be necessary; metabolic and acid-base status should be stable and not require frequent laboratory analysis or change in medications. Infections must be at a minimum; the patient's temperature and white blood count should be within normal range; and a need for frequent antibiotic coverage should not be anticipated. Nutritional status should be optimized, either with oral or enteral feedings; parenteral nutrition can be done in the home, but it does add to the complexity and cost of care. The patient must have adequate airway protection and be able to cough secretions spontaneously or have tracheal suctioning. Some patients with neuromuscular disease on noninvasive ventilation will need either manually assisted coughing or the use of mechanical devices to aid coughing.[24] If the patient has an artificial airway, it must be a tracheostomy not an endotracheal tube. In order for home care to be successful and cost effective, medical, nursing, and respiratory interventions should be minimal and readmission to the hospital not anticipated for at least a month.

Table 6. Patient Characteristics that May Determine Success in Home Ventilator Care*

Ideal	Acceptable	Unacceptable
Individual Coping Style		
Optimistic Motivated Resourceful Flexible Adaptable Sense of humor Directive	Optimistic Motivated Sense of humor	None
Support Systems		
Close family & social supports	Social supports	Lack of family & social supports
Education		
College degree Ability to learn	Ability to learn Mechanically astute	Altered mental status Unable to learn
Financial Resources		
Adequate personal assets Optimal health insurance	Adequate health insurance	Lack of personal assets Lack of health insurance
Medical Condition		
Stable neuromuscular disease Adequate free time off ventilator No other illnesses	Stable neuromusuclar or obstructive disease Limited or no time off ventilator	Medically unstable
Self-Care Ability		
Able to provide self-care and/or direct others	Able to provide self-care	Unable to care for self or direct others

*From Reference 20, with permission.

Patient Characteristics & Psychosocial Aspects of Patient Selection

The medical condition of the patient and the degree of stability play an important role in successful outcome, but many other factors also play an integral role in the success or failure of home care[20] (Table 6[20]). These factors have been discussed at length in the past.[20,25] A psychosocial consult is important for these complex patients and should be initiated as soon as the patient is deemed to be a candidate for home care. The patient may be an excellent candidate, but without family or other social supports or sufficient insurance discharge to home may not be possible.

Predictors of Successful Outcomes in Home Ventilator Care

As healthcare professionals we have an obligation to weigh the pros and cons of home ventilator care for each patient and base the decision not only on the need to move patients out of the ICU but also on the appropriateness of each patient for home ventilator care. One approach to decision making is to develop a scoring system similar to the ones used by many intensivists to determine outcomes of critically ill patients, such as the APACHE II or III scores.[2,3] The APACHE system correlates acute physiology, age, and chronic health data to determine the appropriateness of critical care and patient survival; but for chronic ventilator patients that data would not be adequate. The determination of successful outcome depends on the predictors listed in Table 7. Each factor needs to be weighted according to the importance it may have in determining success. Factors less conducive to successful home care are given a heavier weight. For example, primary lung diseases would have a heavier weight than some neuromuscular diseases based on the experience with prior patients. ALS may have a heavier weight than other neuromuscular diseases because it usually progresses much more rapidly and may involve an older population.

Table 7. Predictors of Successful Outcomes for Home Ventilator Care

Primary diagnosis
Reason for ventilation
Comorbidity
Mode of ventilation
Ventilator settings
Hours/day of ventilation
Physiology
Nutrition
Degree of dyspnea
Functional capability
Age
Cognitive status
Family support
Financial resources
Coping style
Communication ability
Aspiration potential

The presence of other chronic diseases (especially those that limit survival, decrease function, require frequent medical interventions, or place the patient at high risk in the home environment) are given a heavier weight. Age plays a significant role, especially when coupled with the other predictive values; age over 60 years carries a heavier weight. Physiologic data are to be weighted according to the values; pH and P_{aCO_2} are assessed together to monitor compensation for acid-base abnormalities. A P_{aO_2} of 60 torr on room air carries less weight than the same P_{aO_2} on an F_{IO_2} of 0.40. Serum albumin as an indicator of nutritional status is weighted. Hemoglobin values are important because of oxygen carrying capacity.

If the patient is on noninvasive ventilation by a negative pressure generator or positive pressure via a nasal or oral interface, a lower weight would be used because these patients may have some capacity for spontaneous breathing and sustain fewer complications than those with an artificial airway. Patients on noninvasive ventilation may have less potential for aspiration because patients at high risk may be more likely to have had a tracheotomy to bypass the upper airway. The ventilator settings (specifically, the F_{IO_2}, need for PEEP, rate, and volume) can be rated—heavier weights are assigned for high F_{IO_2}, PEEP, and a high minute ventilation because of the increased complexity and potential instability. Heavier weights are also applied when > 16 hours/day of ventilation is required because these patients are at higher risk in the event of ventilator or power failure. The patient who continues to experience dyspnea on optimal ventilator settings may have a poor prognosis compared to patients who have no complaints of dyspnea. The Borg or some other visual analog scale can be used for this assessment.

The cognitive status of the patient plays a major role in decisions about home care. A patient who can make informed decisions, learn the necessary care, and direct care does much better in the home setting than the patient with fluctuating cognitive status, inability to learn or to make decisions, and the inability to direct others in his care. Thus, cognitive dysfunction may carry a heavier weight than the primary disease because it would be impossible to send a patient home unless full-time caretakers were available. The patient who can either provide all of his own care or direct others is a good candidate for home care. Family or other social supports also play a significant role. One study showed that a patient's residing with family prior to hospitalization was a major determinant of successful home care.[26] The investigators looked at a cohort of 44 patients admitted to a respiratory unit for weaning; of these patients, 29 (66%) could not be weaned and 22 (76%) of those were discharged to home. When the family is fully capable, both physically and mentally, of providing full care or assisting with care, discharge to home is more realistic. The patient's coping style has a major impact on the feasibility for home care; as noted previously the patient who is optimistic, motivated, resourceful, adaptable, and flexible is a much better candidate for long-term care in the home. The patient who does not have any of these internal strengths will do very poorly. Finally, the patient who can communicate his needs verbally has a much better sense of control over his life than the patient who is unable to communicate at all. All of these factors must be looked at together in order to make a reasonable and safe decision about long-term ventilator care. Selecting the appropriate time for determining many of the predictors plays a role in the decision. Some of the physiologic data may change once the patient is stabilized, and more than one determination may need to be made. If possible, these factors should be assessed prior to placing the patient on the ventilator.

Discharge Planning

Planning the discharge of ventilator-dependent patients requires a team of individuals, including the patient and family, to work together to ensure a safe transition from hospital to home. Make and I[10] describe the discharge process by a 6-step approach that includes

- stabilization of the patient's medical condition,
- evaluation and development of realistic goals,
- rehabilitation planning to set the stage for training,
- rehabilitation training to increase strength and endurance,
- discharge planning to assure success, and
- home care and follow-up to decrease medical complications.

The process used must include patient and family education, selection of caregivers, rehabilitation, selection of home care companies, equipment selection, home assessment, psychosocial support, maximizing free time from the ventilator, improving nutritional status, assessment of swallowing, speech evaluation, artificial airway management, and a smooth transition to the home environment. Many of these issues have been reviewed in previous literature.[10,20,27-36]

Team Concept

The discharge planning team may consist only of the physician, respiratory care practitioner, nurse, social worker, and patient or it may also include members of the rehabilitation department, such as physical and occupational therapists, dietitian, psychiatrist, speech pathologist, hospital discharge planner, case manager, and members of the home care agencies. Regardless of who is on the team, communication among team members plays a major role in the timely and safe discharge of the patient. The team needs to meet on a regular basis to discuss the progress of the patient, set new short-term goals, and address issues that may be interfering with possible discharge. The patient and/or family may need to be present at all or some meetings and take part in the decision making; if they cannot attend, then one team member needs to communicate to the patient what was discussed at the meeting. Because the physician is ultimately responsible for the patient, he or she may discuss specific issues or decisions with the patient or delegate this task to the discharge planner. It is important not to confuse the patient or family with conflicting information, and it is not always easy to tell patients what they *need* to hear rather than what they want to hear. For example, a patient may be told that he will be home in a few days, the patient interprets that as 2 days but the person telling him really meant about 2 weeks. It is best to be concrete when discussing issues and even put them in writing—leaving no room for misinterpretation.

Some members of the team may be present only when specific concerns are being discussed. The home nursing agency may attend only when the discharge date has been set and specific concerns related to skilled nursing and other outside caretakers are being determined. The HME representative may need to attend most meetings because he will be responsible for equipment and may also teach the patient and family prior to discharge.

Caregiver Selection

For the patient who is totally independent or has the maximal resources to hire any caregiver, the caregiver selection process may not be as important as in other situations because the patient will provide his own care or hire someone to do it. For other patients, this is not the case, and decisions need to be made about provision of care (Table 8). The patient who can provide most of his care will need only minimal help from the family or outside caretakers; patients in this category may include those with diaphragmatic paralysis, kyphoscoliosis, postpolio syndrome, and some patients with COPD. Those who can only perform minimal care will need more help from the family or outside caretakers and may or may not be able to direct others in their care. If they can direct others, that determines the skill level needed by caretakers. Patients in this category may have postpolio syndrome, muscular dystrophy, COPD, or another primary lung disease. Those patients who cannot provide any of their own care will need total help from the family and may or may not be able to direct their care. Patients and families in this category may be younger, but when the family is providing all of the care they may need periodic respite care from outside agencies. This category may include patients with spinal cord injury, muscular dystrophy, ALS, or primary lung disease. Younger patients may have siblings living at home who can participate in daily care; the complexity of what they do will depend on their age and willingness to participate. Other patients may have family members who are willing to participate in the care, but they may not live at the same address. Family members may work out a system with each other to provide care for certain hours of the day or week. In order for this to work, there must be some consistency and dependability among the family members.

When the patient cannot care for himself and family members are unable to provide care because of ill health or work commitments—a situation faced by many patients, especially if the patient and spouse are elderly or the spouse needs to continue working in order to maintain health insurance—the problem may be solved if the patient can qualify for professional or nonprofessional caretakers. The spouse who needs to continue working and must also provide 8 to 16 hours of care a day will need lots of support and close observation for fatigue, anger, inappropriate decision making, inadequate care, or lack of attentiveness to the needs of the pa-

Table 8. Patient and Family Capabilities and the Selection of Caregivers

Patient/Family Capabilities	Care Needs
Patient provides own care	Needs minimal or no help from family
Patient provides only minimal care but can direct others in care	Needs maximal help from family or outside caregivers
Patient cannot perform any self-care	Needs full support from family or caregivers
Family member is healthy and can provide all care	Other family members will take on the responsibility to provide intermittent support
Family member unable to provide any care, secondary to ill health	Will need full-time support from professional or unskilled caretakers
Patient requires complex care	Will need full-time professional care

tient. Some patients need full-time skilled nursing care because of the complexity of care required (such as young children with primary lung disease). Full-time skilled care should not be necessary unless the patient's medical condition is unstable, in which case the patient should not be in the home setting. Many patients who require full-time ventilatory support do have professional nursing support because that is all the insurance company reimburses. Many of these patients could do well with personal care assistants who are hired and trained by the family and/or hospital; some states have special funding for this service, and it is recognized that the use of unskilled caretakers greatly decreases the costs of home care. Lastly, we have patients who require intermittent skilled nursing visits and have home health aides for help with bathing, toilet, and homemaking. The patient may be able to perform some of the ventilator care or suctioning. The home health aide may be able to clean and assemble the ventilator circuits or do chest physical therapy if trained and directed by the patient or family. These issues need to be addressed early in the discharge process, so the appropriate people are taught and outside caregivers are obtained.

Patient & Family Education

A major part of the discharge planning process is education of the patient, family, and other caregivers. The educational program should be tailored to meet the needs of the individual patient. Table 9[20] is a list of basic skills to be learned by the patient, family, or other caregivers. The National Center for Home Mechanical Ventilation has recently developed a list of educational objectives for home care;[37] this list is 40 pages long, which attests to the complexity of discharge planning and education. The team must decide who will do the teaching and how it will progress; generally, teaching should begin with the simple tasks and progress to the more complex. As each task is learned, the patient, family, or caregiver should take over the routine performance of the task in order to become proficient and develop confidence. Documentation of the educational process is mandatory not only for communication to other team members but also for hospital accreditation purposes. Procedural outlines need to be in writing, and all team members should teach the patient from the outline. In this way the patient is taught only one way to do the procedure, decreasing confusion. The patient and family's learning styles should also be assessed and documented to make the educational process smoother. Other factors that may affect the patient and family's ability to learn include their readiness to learn, cognitive functioning, level of fatigue and endurance, acceptance of the disease and the need for ventilation, and emotional stability. If the patient has not accepted the disease, is angry or sad about his situation, or is still experiencing the effects of benzodiazepines or other mood-altering drugs, the learning process will be limited until these situations are reversed. Neuropsychiatric testing may be beneficial for assessing cognitive function or exposing psychiatric disorders that might interfere with the learning process and for determining learning style for the individual patient.

Learning the necessary skills through demonstration at the bedside (without distractions from too many people) was found to be important when one group of patients and families were interviewed.[38] This same group also felt that sessions should be short and that learning skills was more important than learning about lung function. Many patients and families may have learned by observation before the actual educational process begins and may have observed procedures performed with varying techniques, which may interfere with learning the appropriate technique for home.[20,31,39]

Rehabilitation

When patients have had lengthy stays in the critical care unit, some form of rehabilitation will be necessary prior to discharge to the home. After prolonged bedrest, muscle wasting of the extremities may have occurred, and the patient may also have developed contractures and foot drop. The patient's ability to use his hands may be impaired because of immobility, multiple intravenous lines, arterial lines, and restraints. Whether the patient stays in the ICU until discharge or transfers to a stepdown or specialized respiratory unit, a rehabilitation consult is needed. The patient may also be depressed about his medical condition, frustrated and angry about the lack of progress, and not motivated to participate in a rehabilitation program. The program should be re-

Table 9. Skills Needed by Patient and/or Caregivers prior to Discharge*

Self-Care Techniques
- Airway management
  - Tracheostomy and stoma care
  - Cuff care
  - Tracheal suctioning
  - Changing the tracheostomy tube
  - Changing the tracheostomy ties
- Chest physical therapy techniques
  - Percussion
  - Vibration
  - Coughing
- Medication administration
  - Oral
  - Inhaled
- Bed-to-chair transfers
- Feeding-tube care
- Indwelling-catheter care
- Implantable-I.V.-line care
- Bowel care
- Switching from the ventilator to weaning device

Equipment Maintenance
- Ventilator
- Humidifier
- Suction machines
- Battery and charger
- Oxygen administration
- Manual resuscitator
- Troubleshooting for problems
- Cleaning and disinfection

Emergency Measures
- Ventilator failure
- Power failure
- Dislodged tracheostomy tube
- Obstructed airway
- Cuff leaks
- Shortness of breath
- Ventilator circuit problems
- Infection
- Falls
- Bleeding
- Cardiac arrest

*From Reference 20, with permission.

viewed with the patient and an assessment made of the patient's desires. At this point, short-term goals that are easily achievable and realistic should be set. As goals are accomplished, the patient may begin to feel more positive and more motivated to progress to the next step. Increasing the patient's mobility by getting him out of bed and away from the bedside with the use of a portable ventilator can be accomplished only by improving strength and endurance through an exercise program and adequate nutrition. If a portable ventilator is not available, a manual resuscitator may be used to ventilate the patient if he is clinically stable. It is important for the patient to get away from the stationary ventilator, which has been his lifeline; he needs to know that life can continue outside the confines of his room and the 6 feet of ventilator tubing. For many patients, being able to get out of the hospital room gives them an impetus toward a more positive attitude. This, coupled with the progress they are making in the rehabilitation program, may be the first step toward improving quality of life.

Even when patients have underlying neuromuscular disease, improvements in strength and endurance can be accomplished. Patients may need bracing to support their torso and/or neck while in the upright position and splinting to prevent further contractures or loss of function of their hands. If full function is not attainable, the physical and occupational therapists will assess the need for assistive devices such as wheelchairs, walkers, canes, environmental control units, arm supports, and other devices that will help the patient to become more independent. Arm supports or a tray on the wheelchair may allow many patients with limited function to feed themselves and even do their own suctioning. When the patient needs a powered wheelchair, the prescription should be specific and requires the expertise of a therapist and equipment company with much experience in wheelchair modifications. When the patient has a progressive neuromuscular disease, the wheelchair may need a reclining back, head support, or molded seating; these needs should be anticipated and incorporated into the wheelchair prescription so expensive modifications will not need to be made during the next year.[24,40]

The rehabilitation program must be individualized for each patient, with the long-term goal of transferring the patient home with as much function as his disease allows. A patient with COPD may return to full functional independence and be able to perform all of his activities of daily living; whereas, a patient with muscular dystrophy will regain only limited independence in daily activities. As the patient becomes more involved in the rehabilitation process, a daily schedule must be formulated to allow adequate rest periods between activities; otherwise the patient may become overwhelmed during part of the day. All patient activities of daily living, treatments, weaning times, rest periods, and exercise sessions need to be included. A list of the short-term goals and the patient's accomplishments must be placed where the patient and family can see it. As the patient makes gains in the program, no matter how small, positive reinforcement is important.

Weaning from the Ventilator

As the patient progresses through the rehabilitation process, and with improvements in nutritional status, ability to wean may also improve. The patient may also be more psychologically ready to wean. When prior weaning attempts had been initiated in the ICU, the patient may not have been ready physiologically, and the effects of benzodiazepines may still have been present. The patient may also have had prolonged effects from neuromuscular blockade.[42] He may have thought of himself as a failure and essentially given up.

When weaning trials are initiated in the rehabilitation setting, the team has a major goal of helping the patient and family overcome their fear of the weaning process. The procedure and the purpose for weaning must be fully explained to patient and family. They need to understand that different techniques can be used, so if one technique does not work well other options exist. The patient's ability to be off the ventilator is determined prior to the weaning trial. The initial trials of weaning should be done with the critical care ventilator because the options of pressure support, SIMV, and spontaneous breathing are readily available, and the patient's status can be monitored easily. If the patient is able to progress in those weaning trials, then switching him to a weaning mode that is available in the home must be initiated. Patients who have been weaned on pressure support should have the level of pressure support decreased to the minimal level needed to overcome ventilator work of breathing or be placed on a flow-by system prior to conventional T-piece trials. The length of weaning time may decrease without pressure support, but if the patient is to have free time from the ventilator then T-piece weaning must be initiated. The goals of establishing free time include allowing the patient to have some independence from the ventilator and providing a safety mechanism in the event of power or equipment failure. When the patient does well on the T-piece, the next step is to use a fenestrated tracheostomy tube with cuff deflated and place the tracheostomy button in the tube. Prior to doing this, the patient's airway above the cuff site must be assessed for granulation tissue, vocal cord damage, narrow airway, tracheomalacia, and the position of the fenestration within the trachea. Some patients may need laser excision of granulation tissue, others may need a customized fenestrated tracheostomy tube, and still others may be able to breathe only through the upper airway with the use of a one-way valve on the tracheostomy tube, allowing inspiration through the valve and exhalation through the upper airway.

Weaning trials are initially done with the patient at rest, but as the weaning progresses allow the patient to perform some functional tasks—in general, tasks that don't require much energy expenditure, such as washing hands and face or doing tracheostomy care. As the patient tolerates these activities, then other higher level activities can be incorporated into the weaning process. Monitoring the patient is essential during these trials and should consist of pulse oximetry, vital signs, work of breathing, periodic blood gas analysis, and ventilatory mechanics. Prior to discharge, the weaning method must be simplified and performed as it will be done in the home, and the patient and family must be comfortable with the setup. The time periods must be consistent and the patient stable during the period. Guidelines for increasing or decreasing weaning times after discharge should be provided.

Communication Strategies

Patients with a tracheostomy may not be able to communicate effectively with staff or family and may have relied on the ability of others to read their lips and/or used other communication strategies such as writing or a letter board—methods that are inefficient and usually frustrating for the patient and staff. Some of the effects of loss of speech are listed in Table 10. The inability to communicate affects the patient's self-esteem, which in itself affects quality of life. Frustration can lead to anger that may then cause the patient to withdraw and increase dependence on the staff. The patient may fear that his needs will not be met or the staff or family will not respond to him; he may also fear that his voice is lost and that he will never speak again. All of these things can cause more depression and feelings of isolation.[29] Because of these things, a method of communication should be determined early in the hospitalization. The ability to communicate can enhance motivation and give the patient a sense of control over his environment. The speech pathologist should assess the patient and, with input from the patient and staff, determine the appropriate mode of communication.

Table 10. Effects of Loss of Speech

Decrease in self-esteem
Change in quality of life
Frustration
Anger
Withdrawal
Increased dependency
Fear
Depression
Isolation

The type of communication system chosen depends on the patient's respiratory condition, cognitive status, physical condition, motor and sensory limitations, emotional status, and tolerance for optimal position changes. Oral speech methods depend on the patient's ability to articulate. Choice of cuff deflation, a cuffless tracheostomy tube, or a fenestrated tube depends on the patient's ability to handle upper airway secretions, the presence of intact oral and laryngeal musculature, and stable cardiac and pulmonary function. A one-way valve placed inline on the ventilator circuit on the tracheostomy tube may be tried. To use this valve, the cuff must be deflated, the tidal volume increased, and the patient monitored closely for signs of upper airway obstruction. All of the ventilator volume is directed towards the lungs and upper airway, and the patient may speak during both phases of the ventilatory cycle. With any method that involves cuff deflation, the patient needs close monitoring by pulse oximetry, vital signs, blood gas analysis, and assessment for aspiration. These patients may need a barium swallow with videofluoroscopy. If the patient cannot tolerate a cuffless tube or cuff deflation, a 'talking' tracheostomy tube may be tried.[42] The 'Talk Tube' (Portex, Portex Inc, Wilmington MA) is one example of such a tube (Fig. 4). Success with talking tracheostomy tubes is variable and depends on the willingness of the patient to practice, placement of the airflow port within the tracheal lumen, leakage of air through the stoma, vocal cord dysfunction or paralysis, and occlusion of the airflow port with secretions. Because this tube requires a separate compressed gas source, it may not be practical for some patients in the home environment.

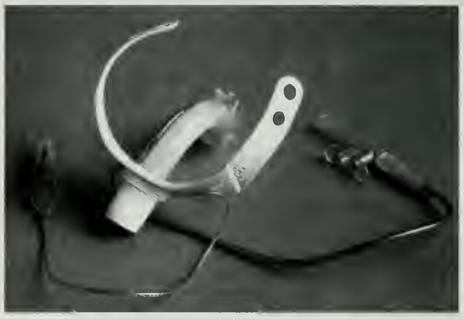

Fig. 4. A 'talking' tracheostomy tube. The small tube separate from the pilot tube terminates just above the cuff; gas is directed through this tube above the cuff and through the vocal cords.

Phonation may also be achieved with the use of an electrolarynx, which attempts to simulate the vibratory role of the larynx. This device is placed either directly on the neck or in the mouth to create sound. The patient must be alert and able to articulate well, follow directions, and integrate new information.[29,43] The electrolarynx can provide an effective means of communication that outweighs the artificial sound of the voice.

Non-oral modes of communication should always be considered when oral methods are not feasible. Some of these systems rely on good hand function, but some utilize infrared light beams, or pointers, and are computerized. When possible, a simple device should be used, but some patients may require more sophisticated devices, which are also more expensive. Some devices may not be covered by the medical insurance and prior approval may be necessary.

Home Care Equipment

The equipment needed for mechanical ventilation in the home varies from patient to patient depending on the mode of mechanical ventilation, patient diagnosis, age of the patient, and patient stability. Patients who are ventilated noninvasively and require only nighttime ventilation will have minimal requirements—a ventilatory assist device, circuits, nasal or oral interface and straps, and for some supplemental oxygen and/or a humidifier. In general, these patients will not need a backup ventilator or any other accessory equipment, and long-term maintenance will be minimal for the patient and home care company. The other extreme is the patient with a tracheostomy and full-time ventilator dependence. Table 11 lists the necessary and potential equipment and supply needs for this patient. The issues related to this type of patient are what is really necessary, the preferences and experience of the respiratory care practitioner, the overuse of complex equipment, the availability of equipment and supplies from the home care company, what the insurance company will reimburse, and, in some instances, what the professional home nursing agency finds necessary. Patients are sometimes sent home with enough equipment to furnish an ICU, which may continue to give the patient and family the impression that a hospital level of care is still needed and prohibit them from attaining a level of independence. If the patient continues to require 'high-tech' monitoring, he is most likely not stable enough to be in the home setting. Experienced respiratory care practitioners and nurses with good clinical skills should not have to rely on ICU level monitoring to make judgments about the patient's status, and family members or other unskilled caretakers cannot be expected to interpret and alter therapy based on data from ICU monitoring equipment.[44]

Table 11. Necessary and Potential Equipment and Supplies Needed for the Patient with a Tracheostomy

Equipment	Supplies
Ventilator(s)*	Water traps*
Manual resuscitator bag*	Tapered 5-inch flex tubes*
Humidifier and thermostat*	Aerosol tubing
Temperature alarm*	Tracheostomy tube strap*
Suction machine* Electrical Battery	15- and 22-mm adapters
Ventilator and battery cable	Reservoir bag
Battery-12 volt	Test lung
Battery charger	Tracheostomy tube (backup)*
Secondary ventilator alarms	Swivel adapters*
Remote alarms	Artificial noses*
Air compressor	Cleaning supplies
Oxygen source	Oxygen connecting tubes*
Ventilator circuits*	Suction connecting tubes*
Exhalation valves*	Suction catheters*
PEEP valves	Bacteria filters*
Power strip—surge protector*	Gloves*
Large volume nebulizer	Luer tip syringe (cuffed tracheostomy tubes)
Oxygen analyzer	Distilled water*
Pulse oximeter	Small volume nebulizer
	MDI spacers
	T-piece or tracheostomy mask

*Necessary.

About 2 weeks before discharge, the home care ventilator and other equipment to be used in the home should be brought into the hospital so that patient and family can become comfortable with the equipment. Many times the family member and/or patient are taught suctioning and nebulizer treatments with the hospital wall vacuum and piped-in compressed gases or they are accustomed to three or four oxygen outlets. If they do not practice with the home care equipment, they will forget to turn on flowmeters or the suction machine and forget to switch the oxygen connecting tubing to the appropriate device. If the insurance company will pay for only one suction machine and the home care company does not want to place a second machine in the home, then a battery-operated machine should be used; with a power failure, the patient can still be suctioned.

A problem associated with not ordering specific name brand supplies is that HME providers may have similar supplies that do not work as effectively. They may have a good price on certain items for a big cost savings, but the potential for problems is increased. For example, swivel adapters for tracheostomy tubes can affect inspiratory pressures because of differences in resistance among brands. Flexible tubes used between the swivel and the exhalation valve are available in different lengths and materials; some of these tubes are so soft they can actually become twisted and occlude the airway. Flexible tubes longer than those used in the hospital between airway opening and circuit Y increase dead space. These may be minor problems but when coupled with other problems in the home the results can be frustrating. Current literature should be reviewed for research on all aspects of care and on the devices being used in the home. The Food and Drug Administration (FDA) alert[45] related to hazards of ventilator humidifiers may have an impact on the home care company's policies and costs if humidifiers must be replaced or a temperature alarm system added. The issue related to humidifiers stresses the importance of proper preventive maintenance and daily monitoring of the equipment.

Home Assessment

The home environment should be assessed prior to discharge when a patient is on life support equipment, has multiple accessory items, and will need a wheelchair or other assistive device for mobility. Patients with long-standing neuromuscular disease may have modified the home already to accommodate a powered wheelchair, but patients who are recently disabled (eg, with spinal cord injury) may need extensive modifications to the home. This process may need to begin early in the hospitalization in order to ensure completion by discharge. For straightforward cases, the assessment can be made by the respiratory care practitioner from the HME company. For more complicated cases, a hospital representative with experience in home care, an occupational therapist, physical therapist, and a home nursing agency may also be involved. The assessment (Table 12[20]) should include accessibility to the home and between rooms, provision to manipulate a manual or motorized wheelchair between rooms and to the outside, and the ability of the patient to sit at a table or counter if wheelchair dependent. The bathroom assessment should include the need for safety bars for the bathtub or commode, the need for a higher commode, and the need for an elevated toilet seat, and the need for a bath chair or hand-held shower nozzle. When modifications are recommended, it must be remembered that they are not covered by health insurance, which means that they are out-of-pocket costs for the family. Therefore, only absolute necessities should be recommended.

Table 12. Assessment of the Ventilator-Dependent Patient's Home*

Accessibility
- In and out of home
- Bathroom
- Kitchen
- Between rooms
- Wheelchair mobility
  - Doorway width
  - Thresholds
  - Stairways
  - Carpeting

Equipment
- Space
- Electrical power supply
  - Amperage
  - Grounded outlets

Environment
- Temperature
- Lighting
- Living space

*From Reference 20, with permission.

Psychosocial Issues Related to Home Ventilator Care

The stresses faced by individuals requiring chronic ventilatory support are the same as those encountered by patients with chronic illness but with the added stress of dependency on a machine for life. Strain[46] described basic psychological stresses faced by anyone with chronic illness, including: (1) threat to the belief that one is capable, independent, and indestructible; (2) separation anxiety; (3) fear of loss of love and approval; (4) fear of loss of control of bodily functions; (5) fear of bodily injury; and (6) reactivation of guilt or shame for past transgressions. When patients are placed on a ventilator and realize that it is needed to support life, their main concern may be centered around a fear of dying.[28] Over time, they may suppress or deny their feelings of anxiety in order to help themselves cope with the day-to-day existence. A certain degree of denial is necessary for survival and may help the patient maintain hope and avoid serious and incapacitating despair.[47] The inability to communicate adequately may further aggravate the sense of isolation and, of itself, make the patient more dependent on the staff and family. If the patient has always made the decisions in the family and now suddenly has to rely on others, his sense of helplessness increases. Family members may also feel increasingly anxious because of the need to make more decisions and may feel angry toward the ill family member for placing them in this position. Establishing a means of communication for the patient is important and should be done as soon as possible. Frequent communication between patient, staff, and family is also important to decrease anxiety and to prevent lapses of communication and the potential for mixed messages. Without frequent communication, patient and family may think that the staff is avoiding them, which increases their anxiety and may engender mistrust.

Many ventilated patients in the ICU may have had a psychiatric evaluation because of anxiety, depression, or delirium and the need for benzodiazepines and/or neuroleptics, but now the evaluation must center around the ability of the patient and family to cope with long-term ventilatory support. The evaluation should include review of the past history of the patient and family, their ability to cope with stressors, recent losses or changes in social situations, past psychiatric history, and the patient's cognitive functioning. Patients with longstanding hypoxemia may have mild-to-moderate degrees of cognitive impairment that may limit their educability and require more repetition and cueing when learning new tasks. Some of these patients may have impairment of higher cognitive functions that involve abstraction and more complex visual motor integration.[48,49] Patients who continue to have delirium and an altered level of consciousness when the acute illness is over should have a full neurologic, infectious, toxic-metabolic (including medications), and cardiac workup to determine causes of the delirium.

Treatment for each patient is individualized; some patients may require ongoing psychotherapy and pharmacologic intervention, and others may benefit from behavioral interventions such as relaxation or biofeedback. All patients should be assured continuity of care because as they progress through the rehabilitation process and gain some independence they may fear abandonment. Symptoms of depression are not uncommon and usually occur in response to various losses associated with the illness. These may include loss of independence, changes in role, changes in physical function, changes in self-image, and fear of rejection by family. When the patient is allowed to mourn the loss and as independence and autonomy increase with rehabilitation, the sense of hope is usually renewed.[28]

Psychosocial issues related to discharge and home care are listed in Table 13. The patient is going from what he considers a safe environment with full medical support to home with limited family support or unfamiliar caretakers, and he may be ambivalent about going home. On discharge, the reality of chronic illness and the need for long-term ventilatory support may set in and depression may return. Some patients have this magical sense that when they go home their life and physical capacity will be back to normal; patients who haven't been able to walk more than 5-10 feet will think that when they get home they will be able to walk throughout the house and perform tasks that they were able to do prior to the admission. There is a loss of privacy, especially if caretakers are constantly present in the home. The family caretakers may feel overwhelmed with new responsibilities and the constant demands of the patient's illness. This may be coupled with the daily stress of family life and inability to plan for the future. The reactions of the

Table 13. Psychosocial Issues Related to Discharge and Home Care

Change in environment
Ambivalence
Change in caregivers
Loss of privacy
Impact of the illness
Change in roles
Demands on family
Inability to plan ahead
Guilt
Decreased socialization
Structural barriers
Daily stress
Feelings of abandonment

Table 14. Reactions of the Patient and Family to Discharge and Home Care

Increased dependency
Anxiety
Family visits less often
Family misses appointments
Unnecessary risk-taking
Failure to make necessary home arrangements
Increased somatic complaints
Anger
Physical and emotional fatigue
Noncompliance
Depression

patient and family to discharge and home care are listed in Table 14. As the discharge date becomes closer, the patient may become more dependent and develop more somatic complaints. Anxiety may increase, and they may display anger towards the staff as they struggle with feelings of abandonment. The family may struggle with feelings of ambivalence by visiting less frequently, missing appointments for teaching, and failing to make necessary home arrangements. After discharge, the patient and family may become physically and emotionally fatigued because of the added responsibilities and the realization of the chronicity of the illness. The patient may become more noncompliant with the treatment plan and even take unnecessary risks such as poorly planned trips or increased time off the ventilator. The family may display anger at the patient for the demands placed on them and their loss of independence, and the patient may become angry because of the predicament that he and the family are in. These feelings generate guilt that will increase anxiety and depression. The staff can ease the transition from hospital to home by anticipating these problems and meeting regularly with the patient and family to keep them informed of all aspects of the discharge. Discussions about home care must be realistic and should center around solving problems that may develop. Positive feedback to the patient and family as they accomplish all of the necessary tasks is important. Trips away from the bedside and the hospital will help the patient and family gain confidence in their ability to be more independent of the staff. Having them speak with other patients and families who have been through the process is beneficial, and many families will network with other ventilator-dependent family members after discharge. When patients and families interact with other families and are able to problem solve with them, it gives them a tremendous sense of accomplishment and a purpose to life. The patient and family should have met the home-care providers prior to going home. Meeting the home-care coordinator is not enough, they must also meet the therapist and nurse who will be following them. Adequate financial resources must be assured because lack of funding greatly increases the stress to the family. Continuity of care after discharge can be maintained with follow-up visits to the home and then to the clinic or doctor's office. The coordinator should be watchful for signs of increased anxiety and depression, such as increases in demands on the family, increase in number of telephone calls to the healthcare providers, increase in somatic complaints, lack of attention to detail, and a decrease in self-care management. Particular attention must be paid early to detect and intervene if suicidal ideation develops (either active or passive). The psychosocial needs of the patient and family must be continually assessed and support provided for them either through continued psychosocial counseling and/or the provision of respite care. Success in home care is related to a number of variables, including continued effective communication between the patient, family, and healthcare providers; effective coping styles that were present before the illness, and the ability of the family and individual to maintain their identities. Success also depends on the family's continuing to have a family life, with the ventilator-assisted individual partaking in some of the responsibilities for family matters. A strong social support network that includes family, friends, community resources, and the staff is a strong predictor of success.

Home Care Agencies & Community Services

The additional resources that the patient will need depends on underlying condition, age, family resources, and the need for transportation. Patients who require only noninvasive nocturnal ventilation may need no additional services, but the patient on full-time ventilatory support with more complex care needs may require continuous nursing care. Many of these patients may not need professional nursing care, but, due to state licensing regulations and insurance requirements for reimbursement, only registered nurses may provide the care. Many of these patients could easily be cared for by unlicensed personnel as long as they were trained. When a nursing agency is used for registered nurses, the referring hospital must be assured that the nurses have had prior experience with home ventilator care. If the nursing agency does not have staff available, they may advertise for nurses with ventilator experience. The nurses who apply may have cared for ICU patients on a ventilator, with the actual ventilator care performed by a respiratory care practitioner. Such nurses will need additional training and should also be scheduled to come into the hospital to work with the patient. Fewer problems may be encountered, and patient and family will be assured that the nurse knows their care.

The nursing agency may provide only intermittent nursing visits depending on the care needs of the patient and insurance coverage. To continue these visits, the patient must require skilled nursing care on a continuing basis. The need for tracheostomy or gastrostomy tube changes or hyperalimentation may qualify the patient for long-term intermittent visits. The patient may have a home health aide who can provide personal care, meal preparation, and some procedures such as postural drainage but who cannot perform tracheal suctioning. The aide may be able to give medications under the direction of the patient or family. If a home health aide is used, then intermittent visits by the nurse will provide continued supervision of the health aide. Other services such as physical therapy, occupational therapy, social service, and speech therapy may be provided by the nursing agency. When a nursing agency is to be used, it should be contacted as early as possible to ensure coordination of services with the hospital, family, insurance company, and the HME provider.

Other services such as Meals on Wheels, homemaking, and transportation may be needed on an individual basis but may not be readily available in all communities. Some communities or church groups may have volunteer services that provide some respite for the family by visiting with the patient. At discharge, letters should be sent to the utility and telephone companies to inform them of the patient's being discharged on life support equipment. It may be helpful to notify the fire or police department because they may be able to provide emergency transportation services or emergency generator service in case of power failure.

In Conclusion

The transition of the patient from the ICU to home is a complex process that requires careful planning, a dedicated healthcare team, expertise in home ventilator care, cooperation of the insurance company, adequate funding, availability of appropriate equipment and supplies, adequate communication, a safe environment, and a patient and family willing and able to take the responsibility for self-care management at home. If all of these factors are not in place, the discharge process will be laden with problems and a smooth transition to home will be impossible. The patient and family will become distrustful of the team, which will increase anxiety and frustration. It may take weeks for the problems to be cleared and a sense of normalcy re-established.

REFERENCES

1. Knaus WA. Prognosis with mechanical ventilation: the influence of disease, severity, of disease, age, and chronic health status on survival from acute illness. Am Rev Respir Dis 1989;140(2, Part 2):S8-S13.
2. Kruse JA, Carlso RW. Severity of illness scoring: East meets West. Crit Care Med 1993;21:647-649.
3. Knaus WA, Wagner DP, Draper EA, Zimmerman JE, Bergner M, Bastos PG, et al. The APACHE III prognostic system: risk prediction of hospital mortality for critically ill hospitalized adults. Chest 1991;100(6):1619-1636.
4. Robert D, Gérard M, Leger P, Buffat J, Jennequin J, Holzapfel L, et al. Permanent mechanical ventilation at home via a tracheotomy in chronic respiratory insufficiency. Rev Fr Mal Respir 1983;11(6):923-936.
5. Czorniak MA, Gilmartin ME, Make BJ. Home mechanical ventilation: clinical course of patients with neuromuscular disease (NMD) and chronic obstructive pulmonary disease (COPD) (abstract). Am Rev Respir Dis 1987;135(4, Part 2):A194.
6. Make BJ, Gilmartin ME. Care of the ventilator-assisted individuals in the home and alternative community sites. In: Burton GG, Hodgkin JE, Ward JJ, eds. Respiratory care: a guide to clinical practice. Philadelphia: JB Lippincott, 1991:669-690.
7. Plummer AL, O'Donohue WJ Jr, Petty TL. Consensus conference on problems in home mechanical ventilation. Am Rev Respir Dis 1989;140(2):555-560.
8. Criner GJ, Kreimer DT, Pidlaoan L. Patient outcome following prolonged mechanical ventilation via tracheostomy (abstract). Am Rev Respir Dis 1993;147(4, Part 2):A874.
9. Gracey DR, Viggiano RW, Naessens JM, Hubmayr RD, Silverstein MD, Koenig GE. Outcomes of patients admitted to a chronic ventilator-dependent unit in an acute-care hospital. Mayo Clin Proc 1992;67(2):131-136.
10. Make BJ, Gilmartin ME. Rehabilitation of ventilator-assisted individuals. Semin Respir Med 1993;14:530-547.
11. Adams AB, Whitman J, Marcy T. Surveys of long-term ventilatory support in Minnesota: 1986 and 1992. Chest 1993;103(5):1463-1469.
12. American Association For Respiratory Care and The Gallup Organization. A study of chronic ventilator patients in the hospital. Dallas TX: AARC, 1991.

13. Gilmartin ME. Mechanical ventilation in the home: an overview. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott. Probl Respir Care 1988; 1(2):155-166.
14. Splaingard ML, Frates RC Jr, Harrison GM, Carter RE, Jefferson LS. Home positive-pressure ventilation: twenty years' experience. Chest 1983;84(4):376-382.
15. Splaingard ML, Frates RC Jr, Jefferson LS, Rosen CL, Harrison GM. Home negative pressure ventilation: report of 20 years of experience in patients with neuromuscular disease. Arch Phys Med Rehabil 1985;66(4):239-242.
16. Garay SM, Turino GM, Goldring RM. Sustained reversal of chronic hypercapnia in patients with alveolar hypoventilation syndromes: long-term maintenance with noninvasive nocturnal mechanical ventilation. Am J Med 1981;70(2):269-274.
17. Bach JR, Alba AS, Bohatiuk G, Saporito L, Lee M. Mouth intermittent positive pressure ventilation in the management of postpolio respiratory insufficiency. Chest 1987;91(6):859-864.
18. Baydur A, Gilgoff I, Prentice W, Carlson M, Fischer DA. Decline in respiratory function and experience with long-term assisted ventilation in advanced Duchenne's muscular dystrophy. Chest 1990;97(4):884-889.
19. Fischer DA, Prentice WS. Feasibility of home care for certain respiratory-dependent restrictive or obstructive lung disease patients. Chest 1982;82(6):739-743.
20. Gilmartin ME. Long term mechanical ventilation: patient selection and discharge planning. Respir Care 1991;36(3):205-216.
21. Silverstein MD, Stocking CB, Antel JP, Beckwith J, Roos RP, Siegler M. Amyotrophic lateral sclerosis and life-sustaining therapy: patients' desires for information, participation in decision making, and life-sustaining therapy. Mayo Clin Proc 1991;66(9):906-913.
22. O'Donohue WJ Jr, Giovannoni RM, Goldberg AI, Keens TG, Make BJ, Plummer AL, et al. Long-term mechanical ventilation: guidelines for management in the home and at alternate community sites: report of the Ad Hoc Committee, Respiratory Care Section, American College of Chest Physicians. Chest 1986;90(1, Suppl):1S-37S.
23. Kacmarek RM, Stanek KS, McMahon KM, Wilson RS. Imposed work of breathing during synchronized intermittent mandatory ventilation provided by five home care ventilators. Respir Care 1990;35(5):405-414.
24. Bach JR, Alba AS, Bodofsky E, Curran FJ, Schultheiss M. Glossopharyngeal breathing and noninvasive aids in the management of post-polio respiratory insufficiency. Birth Defects 1987;23(4):99-113.
25. Gilmartin ME. Long-term mechanical ventilation outside the hospital. In: Pierson DJ, Kacmarek RM, eds. Foundations of respiratory care. New York: Churchill Livingstone, 1992:1185-1204.
26. Gottlieb DJ, Celli BR. Factors that predict successful weaning and home discharge after prolonged mechanical ventilation. Am Rev Respir Dis 1993;147(4, Part 2): A985.
27. Prentice WS. Transition from hospital to home. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott, Probl Respir Care 1988;1(2):175-191.
28. LaFond L, Horner J. Psychosocial issues related to long-term ventilatory support. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott, Probl Respir Care 1988;1(2):241-256.
29. Weisinger W, Goldsmith T. Artificial ventilation: its impact on communication and swallowing. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott, Probl Respir Care 1988;1(2):204-216.
30. Wilson DK, Kaplan RM, Timms RM, Dawson A. Acute effects of oxygen treatment upon information processing in hypoxemic COPD patients. Chest 1985;88(2):239-243.
31. Gilmartin ME, Make BJ. Mechanical ventilation in the home: a new mandate. Respir Care 1986;31(5):406-412.
32. Gilmartin ME, Make BJ. Home care of the ventilator-dependent person. Respir Care 1983;28(11):1490-1497.
33. Make BJ, Gilmartin ME. Rehabilitation and home care for ventilator-assisted individuals. Clin Chest Med 1986; 7(4):679-691.
34. Feldman J, Tuteur PG. Mechanical ventilation: from hospital intensive care to home. Heart Lung 1982;11(2): 162-165.
35. Make BJ. Long-term management of ventilator-assisted individuals: the Boston University experience. Respir Care 1986;31(4):303-310.
36. O'Donnell C, Gilmartin ME. Home mechanical ventilators and accessory equipment. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott, Probl Respir Care 1988;1(2):217-240.
37. National Center for Home Mechanical Ventilation. Objectives for home mechanical ventilation. Denver: National Center for Home Mechanical Ventilation, 1993.
38. Thompson CL, Richmond M. Teaching home care for ventilator-dependent patients: the patients' perception. Heart Lung 1990;19(1):79-83.
39. Gilmartin ME. Pulmonary rehabilitation: patient and family education. Clin Chest Med 1986;7(4):619-627.
40. Bach JR. New approaches in the rehabilitation of the traumatic high level quadriplegic. Am J Phys Med Rehabil 1991;70(1):13-19.
41. Mathewson HS. Prolonged neuromuscular blockade. Respir Care 1993;38(5):522-524.
42. Wilson DJ. Airway management of the ventilator-assisted individual. In: Gilmartin ME, Make BJ, eds. Mechanical ventilation in the home: issues for health care providers. Philadelphia: JB Lippincott, Probl Respir Care 1988;1(2):192-203.
43. Adler JJ, Zeides J. Evaluation of the electrolarynx in the short-term hospital setting. Chest 1986;89(3):407-409.
44. Gilmartin ME. Monitoring in the home and the outpatient setting. In: Kacmarek RM, Hess D, Stoller JK, eds.

Monitoring in respiratory care. St Louis: Mosby, 1993: 767-787.

45. FDA safety alert: hazards of volume ventilators and heated humidifiers. AARC Times 1993;17(11):23.
46. Strain SS. Psychological interventions in medical practice. New York: Appleton-Century-Crofts, 1978.
47. Gipson WT. Mechanical ventilation in the home: the role of the psychiatrist. Cleve Clin Q 1985;52(3):313-315.
48. Fix AJ, Golden CJ, Daughton D, Kass I, Bell CW. Neuropsychological deficits among patients with chronic obstructive pulmonary disease. Int J Neurosci 1982; 16(2):99-105.
49. Grant I, Heaton RK, McSweeney AJ, Adams KM, Timms RM. Neuropsychologic findings in hypoxemic chronic obstructive pulmonary disease. Arch Intern Med 1982;142(8):1470-1476.

Gilmartin Discussion

Celli: I have a comment, Mary, because I may have a little bit of an answer for some of your questions. Dan Gottlieb at our place decided to look at your data. He looked at about 42 variables in those patients that you admitted to the RCC at the Boston University Hospital. The first outcome he was looking at was whether we could predict in chronically ventilated patients who would be able to be weaned? The result turned out to be 40%. The second outcome was who could go home? For predictions for outcome, he did logistic regression analysis. For the model that included $\dot{V}_E$ and P_{Imax}, minute ventilation and strength came out with a sensitivity and a positive predictive factor close to 90%. Perhaps making some index of that would be useful, at the moment you decide somebody is stable enough to consider weaning. When he looked at those that predicted who was going to go home (and by the way, age, nutrition, all those things were looked at), the only factor that was predictive was the presence of a family. He presented it last year at the ATS.[1] He's writing it up to send it out. This was not validated prospectively, which is the problem, but it may form the basis of prospective validation. I'll be very happy to have Dan send you a copy of his work.

REFERENCE

1. Gottlieb DJ, Celli BR. Factors that predict successful weaning and home discharge after prolonged mechanical ventilation (abstract). Am Rev Respir Dis 1993;147(4, Part 2):A985.

Gilmartin: Yes, that would be great.

Bach: You have a negative bias against using ventilatory support for ALS patients, I understand that. Most people do have a negative bias. I probably see more patients with ALS than any other diagnosis. There are a number of these patients who have a very optimistic, positive attitude, even when dependent on a ventilator 24 hours/day. It may be difficult for us (myself included) to understand, but that is, in fact, the case. The other thing is that as these patients get weaker, there are two problems—there's the ventilatory problem and there's the bulbar problem. A significant percentage of these patients with some bulbar function can be managed for 2 years or more by up to 24 hour/day noninvasive ventilatory support.[1] The advantage of introducing noninvasive IPPV to these patients is that if the day comes when the bulbar muscles are severely dysfunctional and you either have to die or accept a tracheostomy tube, you know what it's like to be dependent on a ventilator. In the literature you see complaints by patients and their families of not being properly counseled.[2] They are suddenly in distress, intubated, then told that they have to have a trach; there's no other alternative. They acquiesce and then complain that they were never counseled. One way to get around that is to manage them with noninvasive aids.

REFERENCES

1. Bach JR. Amyotrophic lateral sclerosis: communication status and survival with ventilatory support. Am J Phys Med Rehabil 1993;72(6):343-349.
2. Sivak ED, Gipson WT, Hanson MR. Long-term management of respiratory failure in amyotrophic lateral sclerosis. Ann Neurol 1982;12(1):18-23.

Gilmartin: I think it's helpful if they don't have bulbar problems, but most of the patients that I've dealt with have significant problems with swallowing and aspiration, and they can't talk. I am biased because even the patients I've seen who could do well at home, their families can't take care of them because of their age or the amount of care that is required. The patient may be a great candidate and optimistic, but family can't take care of them, and they don't have insurance to help them out.

Bach: Do you use effective means of augmenting communication—like computerized voice synthesizers? I have patients who are lawyers, judges, and of course there's Charlie Wiedermeyer, the football coach, who's still out there coaching football.

Gilmartin: Jacob Javits also had ALS and continued working.

Bach: Because there are exceptions, we shouldn't condemn everybody.

Gilmartin: No, I don't. That's why I think it's very individualized. I'm looking at all of these other factors. I had a patient with ALS when I worked at the VA in Boston who every day would ask me to kill him.

Hill: Yeah, you don't want to be unrealistically optimistic, either.

Gilmartin: Right. Because it's a terrible disease. It's much more progressive than others.

Sortor-Leger: In regard to patient education, you listed a lot of items used for rehabilitating patients, but I didn't hear you mention teaching energy conservation skills. Do you include this?

Gilmartin: Oh, yes.

Sortor-Leger: Do you teach all of your patients who are going to go home on ventilation?

Gilmartin: I consider that part of their functional adaptation.

Sorter-Leger: Do you personally teach or do you have somebody else who teaches for you?

Gilmartin: That depends on what hospital. At the last place I worked in Boston, we had occupational therapists who did it. In my hospital in Denver, a lot of them don't understand neuromuscular disease. The hospital primarily gets COPD and asthma patients. They're not used to chronic ventilator patients, and they don't have the knowledge to teach a lot of that. But, I agree; I think it's very important.

Hoffman: I support Mary's conclusion about home mechanical ventilation for patients with ALS. My experience has been that it may work initially but that there are substantial long-term problems. As the disease progresses, it's very hard on the family.

Fletcher: If I might echo those same thoughts, I see very few neuromuscular patients at the VA, with the exception of ALS. I've had enough of them, over time. But I think we have a new tool now, that I know that I didn't have 5 years ago: Texas has passed laws allowing you to shut off ventilators in cognitive patients, provided that the patient so wishes. I think we have to look at that. Now, if you put the patient on the ventilator for 3 months and he finds out what life is going to be like, we now have the tool to carry out that man's wishes and his family's wishes—should he not like that kind of life.

Gilmartin: We have done that. We had a patient who was a postpolio patient who developed ALS. He had been a very functional man with his postpolio syndrome. He didn't realize that he could make that decision. A year and a half later, we brought him back to the hospital after he had talked with his minister, his family, the social worker, the full support structure. He decided, "I do not want this anymore." His own physician would not do it, so we brought him back to our hospital and took him off the ventilator. His family was there. It was a good experience. It was difficult for the nurses because he looked very good when he came back. I said, "But why did he look good?" He looked good because he had made a good decision, and he was comfortable with it and happy. He looked relaxed. And he died very pleasantly. So, I agree. I think that we have to make sure patients realize they have that option.

Make: I think there's doubt on both sides of the ALS issue. And it is a very difficult issue. Tony Oppenheimer in Southern California has presented and published some information about ALS-patient perception of their quality of life on a ventilator.[1] These were patients who were receiving ventilation. He found that, by and large, the patients felt they had a reasonable quality of life, and were very accepting of their condition and the fact that they were on the ventilator. That may be asking the question too late. In our experience with ALS patients who are not yet ventilated, they rarely choose to electively initiate mechanical ventilation.

REFERENCE

1. Oppenheimer EA. Amyotrophic lateral sclerosis. Eur Respir Rev 1992;2: 323-329.

Bach: But at the last minute they change their minds, almost invariably.

Make: Patients have the right to withhold and withdraw ventilatory support and to change this decision about life supporting therapies.[1]

REFERENCE

1. American Thoracic Society. Withholding and withdrawing life-sustaining therapy (statement). Am Rev Respir Dis 1991;144(3, Part 1):726-731.

Sortor-Leger: I worked in a clinic in Dallas where the medical director absolutely was totally against ventilating ALS patients. He ventilated none of them. This was a really frustrating experience for me in that a very small but definite percentage of these patients presented with primary diaphragm paralysis prior to having any airway problems and prior to having complete physical paralysis. In my opinion, these patients could have benefitted from noninvasive ventilation had it been offered. I think some real caution should be exercised in regard to having extreme attitudes one way or the other, and I think an individualized approach is really important. I watched this physician discuss ventilation with these patients, and if he had discussed it with me the way he discussed it with them, I guarantee you I would never choose to be ventilated. It became obvious to me how we can absolutely bias patients about an issue we ourselves feel strongly about. That was a growing experience for me to see how much this physician negatively affected his patients' decisions. It helped me to re-evaluate my own thoughts and approach when discussing the opposite viewpoint. It happens all over the country, I'm sure all over the world. I think it's absolutely wrong.

Gilmartin: With muscular dystrophy patients, it's the same problem. Many physicians don't even discuss the options.

Sorter-Leger: It's exactly the same.

Bach: Yes, the majority are biased against it. And some of their responses are absolutely incredible. When we asked them why they didn't even discuss ventilatory assistance, 55% said it was because the patient's quality of life is poor. But they all greatly underestimate what the patients think about their own quality of life. Some of the comments were "Well, the patients don't ask us about mechanical ventilation." Ridiculous comments like that. As if the patients are supposed to know what to do![1]

REFERENCE

1. Bach JR. Ventilator use by muscular dystrophy association patients: an update. Arch Phys Med Rehabil 1992; 73(2):179-183.

Gilmartin: You know, we did have one patient with ALS who was only in his 30s, could only move his eyebrows, and used the computer-assisted communications system. His quality of life, to him, was excellent. In fact, he was a divorced man with custody of his two children, who married his caretaker while he was in the ICU in Boston. I remember the day when I said to him, "I'm going to leave you and your wife alone for the night." We couldn't really send them home on an overnight trial basis because they lived in a trailer up in New Hampshire, so we left them alone in their hospital room, and I never saw anyone so excited when I told him that no one would go into his room unless they really called for someone. So, he had tremendous quality of life.

Gilmartin: I try not to be biased about it. I just have had bad experiences. We had a patient who I know killed himself at home. His wife got up in the morning, and he was disconnected from the ventilator but had occluded the tubing so it wouldn't alarm. He had good use of his hands but couldn't speak or swallow; he was very depressed.

Hill: There is no question that therapy has to be individualized, and what Mary said about coping skills certainly applies here.

Gilmartin: I think it is very important to look at how they coped in the past with problems, and how they got through stressful situations.

Hill: I want to ask a question about your prediction scale, which I think is a very intriguing idea. I was thinking about applying it to the kinds of patients I see in a step-down respiratory unit, consisting of a lot of chronic ventilator patients. I want to know how you account for the time factor? A lot of these patients are coming out of an ICU or are recovering from ARDS or sepsis. They're malnourished and deconditioned, and they score terribly on medical condition and nutrition scales. But after a few weeks, they're better. Eventually, if they haven't weaned entirely, their condition looks much more favorable than initially. How does your scale account for this?

Gilmartin: If you are looking at something that is fairly reversible or you hope it is, then you would still be looking at those social types of issues that may really make your decision for you. I think if they don't have a family or if you don't think that they could do self-care, then that's going to play an opposite role. I think you have to look at it. I don't know what the right timing is. We're looking at a lot of these issues. I think you really need your psychologist's help with a lot of these patients early on, too.

Leger: I have a question about how, financially, you manage the technical help for the patient—for example, all the electronic devices to help speak or use a computer or manage environmental controls? In France, we have little financial support for these such items which, in my opinion, can really improve a patient's quality of life. Unfortunately for us patients must depend on their own money and their own abilities to organize funding for such items, frequently leaving them without such things and a quality of life for many that is questionable.

Gilmartin: In the United States it is variable. There are some funding options for communication devices for some patients, I think, like the Crippled Children's Society. Sometimes they have funded communication devices for some patients. I think it really does vary. Some of the polio patients have had funding from some of these special funds. Medicaid has funded some patients for communication devices. Medicare doesn't.

Bach: Private insurance does.

Gilmartin: And private insurance may. So, it is very variable, even for our patients and some are private pay. That's the problem—when you don't have anything that will fund the communication device, you don't get one.

Sortor-Leger: In my experience in Texas, the ability to secure financing for these additional items has worsened over the last 10 years. As the cost of healthcare has increased, many changes in insurance coverage have occurred. More and more often only essential care items are covered.

Fletcher: I would like to raise an issue for a brief discussion. It might be appropriate near the end of this discussion. We have three major organ system failures that physicians are faced with. One is renal, and we have dialysis that can be done at a fixed expense, in end-stage renal disease, and the patient goes out and comes back, 3 days a week gets dialysis, and can live a reasonable quality of life. We have cardiac failure, and either the patient dies or they get a transplant, so there's a finite limitation to this chronic disease. But with respiratory failure, physicians and caregivers are faced with a disease that can go on for a long period of time, requiring 24-hour ventilation, care, monitoring—probably the potentially most expensive chronic disease that we're seeing. And, we're talking about the responsibility we have to the patient to provide care and let them make the decision? Does anyone here feel that we have a responsibility to society in general, and to medicine, to limit the cost of the care these patients receive and make the tough decisions that, if we don't do so as a group of healthcare providers, somebody else is going to make for us outside of medicine 10 years from now? Because no matter what healthcare reform plan comes along, the costs are going to continue to increase, and sooner or later some decisions about chronic care are going to have to be made.

Stoller: Obviously, it's a very open-ended question. We'll ask John (Bach) for a brief comment because I think we're really not positioned to grapple with that issue, and I think this came up before with regard to the ethics. I think you probably need representation from people other than those who are here, for the sake of this conversation. You want to have representation from patient advocacy groups and ethicists and members of the chronic disease community who are not represented in this forum. To get a balanced view and full consideration might require a whole different Journal Conference.

Fletcher: I don't want this session to close with the fact that we think this is all a given.

Stoller: It's fair enough to raise the question, but I don't think we should aspire to a considered answer—answer is a naive term, obviously—but a considered kind of working through of a complex issue. With that as a proviso, John, do you have some brief comment in that regard?

Bach: All my comments are brief, aren't they? I agree completely with what you said. But there's one thing that I think you should realize: Renting a ventilator and providing mechanical ventilation is one third as expensive as renal dialysis. Renal dialysis disrupts the patient's life far, far more. You have to go to the hospital. You have to be hooked up to a machine for hours at a time during which you can do little else, and you have to do it 3 times a week. When you think about it, it's far more disruptive to both the patient and the family than providing mechanical ventilation.

Fletcher: Than staying in bed next to a ventilator?

Bach: Well, who stays in bed? None of my patients stay in bed, they go to Broadway, they work, they do all sorts of things.

Stoller: I'll take the Chairman's prerogative and perhaps give David (Pierson) the assignment to at least make sure that the final synthesis of this and the discussion tomorrow at least reflect the fact that this is a difficult issue. We just don't have the power to grapple with it here.

Physical Reconditioning of Patients with Respiratory Diseases: Legs, Arms, and Breathing Retraining

Bartolome R Celli MD

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INTRODUCTION

Patients suffering from chronic respiratory diseases decrease their overall physical activity because any form of exercise often results in worsening dyspnea. The progressive deconditioning associated with inactivity, initiates a vicious cycle in which dyspnea increases at ever lower physical demands. With time, patients also adopt a breathing pattern (usually fast and shallow) that is detrimental to overall gas exchange, which may in turn worsen their symptoms. In general, physical reconditioning is a broad therapeutic concept that unfortunately has been equated with simple lower-extremity exercise training. In this paper, I review the current knowledge regarding reconditioning in much broader terms. The effect and role of leg and arm training is critically analyzed, and I make practical recommendations. Because I believe it also to be important, I

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review the concept of breathing retraining in its broad definition.

A word of caution must be raised. The data that form the basis of our current knowledge of reconditioning has been obtained from patients with intrinsic lung disease, such as emphysema, bronchitis, bronchiectasis, cystic fibrosis, and adult respiratory distress syndrome. Little is known about reconditioning in patients with pure 'pump failure,' such as those with degenerative neuromuscular diseases. In these patients, there is every reason to believe that exercise may worsen rather than improve their overall function and sensation of well being. On the other hand, breathing retraining, such as slow, deep breathing, could have an application in such patients as long as extra loads are not placed on already weakened and dysfunctional respiratory muscles. However, patients with symptomatic pump failure, may benefit more from ventilatory assistance and resting than from further training.

PHYSICAL RECONDITIONING

The short- and long-term effects of systematic exercise conditioning have been the subject of extensive investigation. In normal individuals, it is known that participation in a well-designed exercise training program, results in several objective changes: (1) maximal oxygen uptake ($\dot{V}_{O_2max}$) increases, primarily due to increases in blood volume, hemoglobin, and heart stroke volume (although changes in the peripheral utilization of oxygen have not totally been ruled out); (2) specific training produces increases in muscular strength and endurance, primarily, resulting from enlargement of muscle fibers and improved blood and energy supply; (3) muscle coordination improves; (4) body composition changes, with increased muscle mass and loss of adipose tissue; and (5) the subject's sensation of well being improves. In patients with obstruction to airflow, participation in a similar program results in different outcomes depending on the severity of the obstruction. Patients with mild-to-moderate disease, as a rule, manifest the same findings as normal subjects; whereas, as I shall discuss later, patients with severe disease increase exercise endurance and improve their sensation of well being, with little if any increase in the $\dot{V}_{O_2max}$. Little is known regarding outcomes different from the specific effects of training on exercise performance. Similarly, once an effect has been shown, no systematic information documents the effect of maintenance programs on any of the outcomes, including exercise performance.

In patients with chronic obstructive pulmonary disease (COPD), tolerance to exercise is decreased. The most important factors thought to contribute to this exercise limitation are (1) alterations in pulmonary mechanics, (2) abnormal gas exchange, (3) dysfunction of the respiratory muscles, (4) alterations in cardiac performance, (5) malnutrition, and (6) development of dyspnea. Other factors deserve to be mentioned but are less well characterized, including active smoking, abnormal peripheral muscle function, and polycythemia. The most severely affected patients cannot increase exercise to levels at which the training effect is thought to occur (above anaerobic threshold). Fortunately, a large body of evidence supports exercise training as a beneficial therapeutic tool useful in helping these patients achieve their full potential.

Physiologic Adaptation to Training

General Principles

Several principles apply to exercise training, and we must understand them in the context of prescribing exercise to patients with severe pulmonary problems: (1) specificity of training, (2) intensity and duration of the exercise load, and (3) detraining effect.

Specificity of Training

The principle of specificity of training is based on the observations that programs can be tailored to achieve specific goals and that the training of muscles or muscle groups is beneficial only to the targeted trained muscle.

The use of a high-resistance, low-repetition stimulus increases muscle strength (eg, weight lifting), whereas a low-resistance, high-repetition program increases muscle endurance. This specificity of training is achieved by increases in myofibrils in certain muscle fibers (white) for the strength training or increases in number of capillaries and mitochondrial content for endurance training.

The training is specific to the trained muscle. Clausen et al[1] trained the arms and legs of subjects and observed that the decreased heart rate observed for arm-muscle training could not be transferred to the leg group and vice versa. Davies and Sargeant[2] showed that if training was completed for one leg, the beneficial effect could not be transferred to exercise involving the untrained leg. More recently, Belman and Kendregan[3] confirmed these findings, in patients with COPD. They examined the effect of 6 weeks of training in 8 patients who trained only their arms and 7 patients who trained only their legs. They observed improved exercise ability only for the exercise for which the patients trained. Interestingly, they failed to see any changes in muscle enzyme content in biopsies taken before and after the exercise training program.

Intensity and Duration of the Exercise Load

Both intensity and duration of load, profoundly affect the degree of the training effect. Athletes usually train at maximal or near-maximal levels in order to rapidly achieve the desired effects. On the other hand, middle-aged subjects who are not athletes may require less intense exercise. Siegel et al[4] showed that training sessions of 30 minutes about 3 times a week for 15 weeks significantly improved $\dot{V}_{O_2max}$, if heart rate was raised over 80% of predicted maximal rate. In patients with chronic lung disease, the issue of exercise intensity and duration has been studied by different authors, as I review later, but it appears that the greater the number of sessions and the more intense (as a function of maximal performance), the better the results.

In their work, Belman and Kendregan,[3] exercised patients at 30% of maximal exercise capacity, and after 6 weeks of training 4-times/week twice daily during which the load was increased as tolerated, they observed significant improvement in endurance time in 9 of the 15 patients. It is possible that the relatively low training level (30% of maximal) may help explain why 6 of their patients failed to increase the endurance time. In contrast, Niederman et al[5] started the exercise at 50% of maximal cycle-ergometer level, increased its intensity on a weekly basis, and observed endurance improvement in most patients. Other authors[6-9] have used higher starting exercise levels and have achieved higher endurance.

I believe the best study in this regard is that of Casaburi et al,[10] who studied 19 patients with COPD who could achieve anaerobic threshold (moderate COPD with a mean [SD] FEV_1 of 1.8 [0.53] L) before and after random assignment to low-intensity (50% of maximal) or high-intensity (80% of maximal) exercise. The authors showed that the high-intensity training program was more effective than the low-intensity one. They also observed a drop in ventilatory requirement for exercise after training that was proportional to the drop in lactate at a given work rate. Therefore, it seems (1) that training is achieved if the intensity of exercise is at least 50% of maximal and (2) that it can be increased as tolerated.

The number of exercise sessions is also a matter of debate.[3,11,12] As shown in Table 1,[3,11,13] in general as the number of sessions is increased, so is the change in observed endurance time. Because stopping the exercise results in a loss of the training effect, the optimal plan should involve an intense phase and a maintenance phase. I have observed that the maintenance phase is difficult to implement and results in the frequently observed failure to maintain and preserve the beneficial effects achieved through the training.

Table 1. Principal Author, Number of Sessions of Exercise, and Improvement in Exercise Endurance

First Author	Number of Sessions	% Endurance Increase
Belman[3]	48	50
Make[11]	12	12
Epstein[13]	19	30

Detraining Effect

The principle of detraining effect is based on observations that the effect achieved by training is lost after the exercise is stopped. Saltin et al[14] showed that bed rest in normal subjects resulted in a significant decrease in $\dot{V}_{O_2max}$ with 21 days of resting. It took 10 to 50 days for the values to return to those seen before resting. Keens et al[15] examined ventilatory muscle (VM) endurance after training in normal subjects who had undergone VM training. Within 1 month of having stopped training, the subjects had lost the training effect that they had achieved.[15] Therefore, it seems important to continue to train, but the minimum practical and effective timing of maintenance training remains to be determined.

Our exercise program is based on the data and concepts described. Patients are exercised at 70% of the maximal work achieved in a test day. This work is increased on a weekly basis as tolerated by the patient. We aim to complete 24 sessions. This is achieved more quickly if the patient is in the hospital because the sessions are completed on a daily basis. The 24 sessions take longer to complete in outpatients because sessions are held 3 times/week. Each session lasts 30 minutes if tolerated by the patient; otherwise, it is begun as tolerated by the patient, and no further load is provided until the patient can complete the 30 minutes of the session. The person in charge of the training maintains close communication with the rehabilitation planning team.

Lower Extremity Exercise

A number of studies have shown that the inclusion of leg exercise in the training of patients with lung disease is beneficial.[16-20] Cockcroft et al[21] randomized 39 dyspneic patients who were younger than 70 years and not receiving supplemental oxygen. The treatment group spent 16 weeks in a rehabilitation center, where they participated in graduated exercise training; the control group received medical care but was given no special advice to exercise. (The control group served as such for 4 months and was then admitted to the rehabilitation center for 6 weeks. Just like the treated patients, they were instructed to exercise at home following completion of the program. Both groups were similar at baseline.) After rehabilitation, only 2 of the 16 control patients manifested improvement in dyspnea and cough, whereas 16 of the 18 patients included in the treatment group manifested improvement in these symptoms. More importantly, the treated patients showed significant improvement in the 12-minute walk and in the $\dot{V}_{O_{2}max}$ when compared with the controls.[21] In a different setting, Sinclair and Ingram[22] randomized 33 patients with chronic bronchitis and dyspnea to 2 groups. The 17 patients in the treatment group exercised by climbing up and down two 24-cm steps twice daily, with the exercise time increased to tolerance. The patients exercised at home and were evaluated each week by the treatment team. The control group did not exercise but was reassessed after 40 weeks. The degree of airflow obstruction did not change in either group. Similarly, no improvement was observed in strength of the quadriceps, minute ventilation ($\dot{V}_E$), and heart rate. In contrast, results of the 12-minute walk test increased significantly in the patients who were trained.[22] These two studies are particularly important because they were well designed and used randomization in the assignment of patients to the specific treatment groups. More recently, O'Donnell et al[12] compared breathlessness, 6-minute walking distance, and cycle-ergometer work, between two age-matched groups of patients with moderate COPD. The exercise-trained group (n = 23) achieved significant reduction in dyspnea scores and increased the distance walked and the cycle-ergometry work, when compared to the control group (n = 13). This trial is important in that it not only documented increased endurance, but for the first time evaluated the patient's perception of dyspnea, which is the most problematic symptom and the one leading to physical limitation. The results of all 3 studies are summarized in Table 2.[12,21,22]

Table 2. Controlled Studies of Exercise in Patients with Chronic Airflow Obstruction

First Author	n	Duration (weeks)	Results
Cockcroft[21]	16/18 T*	16	↑12 MW, ↑$\dot{V}_{O_{2}max}$
	2/16 C	—	No change
Sinclair[22]	17 T	40	↑FVC, ↑12 MW
	16 C	—	No change
O'Donnell[12]	23 T	8	↑cycling time, ↑12 MW, ↑FVC, ↓dyspnea
	13 C	—	No change

*T = treated; C = controls; 12 MW = 12-minute-walk test; FVC = forced vital capacity; and $\dot{V}_{O_{2}max}$ = peak oxygen uptake.

Numerous studies that have used patients as their own controls have shown similar results, with significantly increased exercise endurance. The mechanism by which this improvement occurs remains a matter of debate. Several studies, including those of Paez et al[20] and Mohsenifar et al,[6] have demonstrated a drop in heart rate at a similar work level, a hall-mark of the training effect for the specific exercise. This is perhaps related to a decrease in exercise lactate level as suggested by Woolf and Suero.[23] More recent evidence in support of a training effect is provided by the study of Casaburi et al.[10] In their group of trained patients with COPD, they showed a reduction in exercise lactic acidosis and ventilation after patients were trained. Furthermore, the reduction was proportional to the intensity of the training. The lactic acidosis rise decreased 12% in patients trained at the low work rate (50% of maximum) and 32% in those trained at the high work rate (80% of maximum). In both groups, heart rate decreased significantly after training.[10] Other studies have failed to document either an increase in $\dot{V}_{O_2max}$ or a decrease in heart rate or lactate at similar work level. I believe that the most important study in this group is the one by Belman and Kendregan,[3] which failed to show a decrease in heart rate at the same work load as represented by the $\dot{V}_{O_2}$. These authors went further and analyzed enzyme content obtained by muscle biopsy before and after training. They observed no change in this parameter. Interestingly, 9 of the treated patients improved their exercise endurance. As stated previously, it is possible that the training effort in this study was too low because training began at 30% of the maximum achieved during testing.[3]

Two recent studies addressed the issue of whether patients with the most severe COPD can undergo exercise training. The reason for the question relates to the fact that patients with severe COPD do not exercise to the intensity required to reach anaerobic threshold or to train the cardiovascular system. Niederman et al[5] exercised 33 patients with different degrees of COPD (FEV_1 range 0.33-3.82 L). When they evaluated the response to training, no correlation was seen between the degree of obstruction and the observed improvement. In other words, those patients with a low FEV_1 were as likely to improve as those with a high FEV_1. Similarly, ZuWallack et al[9] evaluated 50 patients with COPD (FEV_1 range 0.38-3.24 L) before and after exercise training. They observed an inverse relationship between the baseline 12-minute walk distance and $\dot{V}_{O_2max}$ and the improvement. They concluded that patients with poor performance, on either the 12-minute walking distance or maximal exercise test, are not necessarily poor candidates for a pulmonary rehabilitation program. From the data, it seems prudent to conclude that any patient capable of undergoing leg exercise training can benefit from a program that includes leg exercise.

As to the type of exercise training to be prescribed, different studies have used different training techniques. Most studies include walking, both as a measurement of exercise tolerance[24] and as a part of the training program. The classic 12-minute walk during which the distance walked in 12 minutes is recorded is good for patients with moderate-to-severe COPD but may not be taxing enough for patients with a lesser degree of airflow obstruction. We have evaluated stair climbing and have shown that the $\dot{V}_{O_2max}$ can be estimated from the number of steps climbed during a symptom-limited test.[25] Several studies have used treadmill testing and/or step testing even though the training has been done with the patient walking. $\dot{V}_{O_2}$ is higher for stair climbing or treadmill testing than for the more commonly used leg ergometry,[25] presumably because the former uses more body muscles than does leg cycling. Leg ergometry has become popular as a testing device and has been the training apparatus for many recent studies. The ergometer is certainly smaller than the treadmill, and, with the relatively inexpensive units on the market, it is possible to train groups of patients simultaneously.

All the studies cited, relied either on in-hospital or outpatient hospital training. Little information exists regarding implementation of such programs at home. In a unique report, O'Hara et al[26] enrolled 14 patients with moderate COPD (mean [SD] FEV_1 of 1.17 [0.76] L) in a home exercise program. The authors randomized the patients to daily walking while carrying a lightweight backpack (mean [SD] 2.6 [0.5] kg) or the same backpacking regimen with additional weightlifting and limb-strength exercises—wrist curls, arms curls, partial leg squats, calf raises, and supine dumbbell press. The initial load was 4.3 (0.9) kg and was increased weekly by 1.2 (0.5) kg for 6 weeks to reach 10.4 (2.6) kg by the last week. The weight lifters performed 10 repetitions 3 times, avoiding dyspnea, breath-holding, and fatigue, for a total time of 30 minutes daily. Patients documented their exercises in a diary, and healthcare personnel visited the patients each week. After training, all weight lifters had reduced their $\dot{V}_E$ during bicycle ergometry when compared with controls. Furthermore, the weight-trained patients showed a 16% increase in exercise endurance. This study suggests that exercise training can be achieved at home with relatively inexpensive programs, with the beneficial consequence of no hospital visits and in the comfort of the home.

Table 3. Training Method for Leg Exercise Used at St Elizabeth's Hospital, Boston

Train at 60% of maximal work capacity.*
Increase work every 5th session as tolerated.
Monitor dyspnea and heart rate.
Increase work after 20-30 minutes of submaximal targeted work is achieved.
Aim for 24 sessions.

*Work capacity as determined by an exercise test, not necessarily by evaluating heart rate. (See text for discussion.)

In our pulmonary rehabilitation program, we complete testing on an electrically braked ergometer, whereas the training is done on mechanically controlled ergometers. Subjects are tested as outpatients or as inpatients, depending on their condition. Table 3 describes how we train our patients. The program may be tailored to each individual and to the available training equipment.

Upper Extremity Exercise

As stated before, most of our knowledge about exercise conditioning in patients undergoing rehabilitation is derived from programs emphasizing leg training. This is unfortunate because the performance of many everyday tasks requires not only the hands but also the concerted action of other muscle groups that partake in upper-torso and arm positioning. Some of the muscles of the upper torso and shoulder girdle serve a dual function (respiratory and postural). Muscles such as the upper and lower trapezius, latissimus dorsi, serratus anterior, subclavius, and pectoralis minor and major possess a thoracic and an extrathoracic anchoring point. Depending on the anchoring point, they may help position the arms or shoulder or if given an extrathoracic fulcrum (such as fixing the arms in a supported position) they may exert a pulling force on the rib cage. We have shown that in patients with chronic airflow obstruction, as severity worsens, the diaphragm loses its force-generating capacity and the muscles of the rib cage become more important in the generation of inspiratory pressures.[27] When patients perform unsupported arm exercise, some of the shoulder-girdle muscles have to decrease their participation in ventilation, and if the task involves complex purposeful arm movements, the pattern of ventilation may be affected. Tangri and Wolf[28] used a strain gauge to study breathing patterns in 7 patient with COPD while they performed simple activities of daily living such as tying their shoes and brushing their teeth. The patients developed an irregular and rapid pattern of breathing with the arm exercise. After the exercise, the patients breathed faster and deeper, which according to the authors was done to restore the blood gases to normal. We have explored the ventilatory response to unsupported arm exercise and compared it with the response to leg exercise in patients with severe chronic lung disease.[29] Arm exercise resulted in dyssynchronous thoracoabdominal excursion that was not solely due to diaphragmatic fatigue. The dyspnea that was reported by the patients was associated with a dyssynchronous breathing pattern. We concluded that unsupported arm exercise could shift work to the diaphragm and in some way lead to dyssynchrony. To test this hypothesis, we have used pleural pressure (P_{pl}) and gastric pressure (P_g) plots (measured with gastric and esophageal balloons) and evaluated the changes as well as the ventilatory response to unsupported arm exercise and have compared it to leg-cycle ergometry in normal subjects and patients with airflow obstruction.[30,31] We documented increased diaphragmatic pressure excursion with arm exercise and alterations in the pattern of pressure generation, with more contribution by the diaphragm and abdominal muscles of respiration and less contribution by the inspiratory muscles of the rib cage.

Our knowledge of ventilatory response to arm exercise was based on arm-cycle ergometry. It is known that, at a given work load in normal subjects, arm cranking is more demanding than leg cycling as shown by higher $\dot{V}_{O_2}$, $\dot{V}_E$, heart rate, blood pressure, and lactate production.[32-34] At maximal effort, however, $\dot{V}_{O_2}$, $\dot{V}_E$, cardiac output, and lactate levels are lower during arm- than during leg-cycle ergometry.[35,36] Little is known about the metabolic and ventilatory cost of simple arm elevation. Some recent reports underscore the importance of arm position in ventilation. Banzett et al[37] showed that bracing the arms increases the capacity to sustain maximal ventilation when compared to lifting the elbows from the braced position. Others have shown a decrease in the maximum attainable work load and increases in $\dot{V}_{O_2}$ and $\dot{V}_E$ at any given work load when normal subjects exercised with their arms elevated.[38,39] We have recently evaluated the metabolic and respiratory consequence of simple arm elevation in patients with COPD.[27,40] Significant increases in $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ with concomitant increases in heart rate and $\dot{V}_E$ occurred when subjects elevated their arms to 90° in front of them. When ventilatory-muscle recruitment patterns were evaluated by continuous recording of P_g and P_{pl}, the contribution to ventilation of the different muscle groups was seen to shift toward increased diaphragmatic and abdominal muscle use. The observations suggest that if we train the arms to perform more work or if we decrease the ventilatory requirement for the same work, we should improve the patient's capacity to perform activities of daily living.

In several studies both arm and leg training have been utilized; and it has been shown that the addition of arm training results in improved performance and that the improved performance is, for the most part, task specific. In their study, Belman and Kendregan[3] showed a significant increase in arm exercise after exercise training. Lake et al[41] randomized patients to arm exercise, leg exercise, and arms-and-legs exercise. Arm ergometry increased in the arm group, leg ergometry increased in the leg group, and the sensation of well-being improved when both exercises were combined.[41] Ries et al[42] studied the effect of two forms of arm exercise—gravity-resistance and modified proprioceptive neuromuscular facilitation—comparing them with no arm exercise in a group of 45 patients with COPD who were involved in a comprehensive, multidisciplinary pulmonary rehabilitation program. Even though only 20 patients completed the program, they showed improved performance on tests that were specific for the training and reported a decrease in the fatigue in all tests performed.[42] It is worth pointing out that in the study by Keens et al[15] a group of patients with cystic fibrosis underwent upper extremity training consisting of swimming and canoeing for 1.5 hours daily. At the end of 6 weeks, upper extremity endurance had increased, but, more importantly, the increase in maximal sustainable ventilatory capacity was similar to that obtained with VM training. This suggests that VMs could be trained by using an arm-exercise training program.

Because simple arm elevation results in a significant increase in $\dot{V}_E$, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$, we studied 14 patients with COPD before and after an 8-week regimen of 20-minute sessions 3 times weekly of unsupported arm and leg exercise. This was part of a comprehensive rehabilitation program to test whether arm training decreases ventilatory requirement for arm activity. The rise in $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ brought about by arm elevation decreased by 35% and was associated with a significant decrease in $\dot{V}_E$.[43] Because the patients also trained their legs, we could not conclude that the improvement was due to the arm exercise. To answer this question, we have recently completed a study[13] of 26 patients with COPD who were randomized either to unsupported arm training (11 patients) or resistance breathing training (14 patients). After 24 sessions, arm endurance increased only for the unsupported arm training group and not for resistance breathing. Interestingly, maximal inspiratory pressure increased significantly for both groups, indicating that by training the arms, we might be inducing VM training for those muscles of the rib cage that hinge on the shoulder girdle.

Based on the information available, we include arm exercise in our rehabilitation program. As seen in Tables 4 and 5, the methods for supported and unsupported arm exercise, vary in their implementation. Arm ergometry is performed for 20 minutes per session. We start at 60% of the maximal work achieved in the test run, and the work is increased weekly as tolerated. Dyspnea and heart rate are monitored. Maximal work capacity is defined as the watts that the patient is capable of achieving. If the patient's limiting symptom is dyspnea at minimal

Table 4. Training Methods for Supported Arm Exercise Training (Ergometry) Used at St Elizabeth's Hospital, Boston

Train at 60% of maximal work capacity.*
Increase work every 5th session as tolerated.
Monitor dyspnea and heart rate.
Train for as long as tolerated, up to 30 minutes.

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*Work capacity as determined by an exercise test, not necessarily by evaluating heart rate. (See text for discussion.)

Table 5. Method for Unsupported Arm Training Used at St Elizabeth's Hospital, Boston

1. Use dowel (weight = 750 grams).
2. Lift to shoulder level for 2 minutes at a rate equal to breathing rate.
3. Rest for 2 minutes.
4. Repeat sequence as tolerated for up to 32 minutes.
5. Monitor dyspnea and heart rate.
6. Increase weight (by 250 g) every 5th session as tolerated.

work, we exercise him at 60% of the work that makes him stop. In the most severely affected patients, the heart rate is unreliable because they may be tachycardic even at rest and may not show any significant increase with exercise. In these patients, dyspnea may be a more reliable index to follow. In contrast, unsupported arm exercise training is achieved by having the patient lift a dowel (weight 750 grams) to shoulder level at the same rhythm as his breathing rate. The sequence is repeated for 2 minutes with a 2-minute rest period, and the exercises are repeated for 30 minutes. Dyspnea and heart rate are monitored. The load is increased by 250 grams weekly as tolerated. We aim to complete 24 sessions.

Thus, an increasing body of evidence (Table 6[3,13,15,41,42]) suggests that upper extremity exercise training results in improved performance for arm activities, with a drop in the ventilatory requirements for similar upper extremity activities. All this should result in an improvement in the capacity of the patients to perform activities of daily living.

In summary, in this first section I have reviewed the physiologic correlates of exercise training and addressed the specific training of legs and arms. A critical review of the literature indicates that exercise conditioning that includes leg and arm training improves exercise performance and seems to have physiologic explanations different from dyspnea desensitization. An exercise program within the context of pulmonary rehabilitation is attainable by anyone interested and can be a rewarding component of any program.

Table 6. Controlled Studies of Exercise in Patients with Chronic Obstructive Lung Disease

First Author	n	Exercise Focus	Duration	Course (weeks)	Activity	Results
Keens[15]	7	Arms	1.5 hours, once daily	4	Swimming/ canoeing	↑VMT (57%)
	4	VMT	15 minutes, once daily	4	VMT	↑VME (52%)
	4	Control (normal)	15 minutes, once daily	4	VMT	↑VME (22%)
Belman[3]	8	Arm	20 minutes, 4 times/week	6	Arm ergometry	↑arm cycle, no ↑PFT
	7	Legs	20 minutes, 4 times/week	6	Cycle ergometry	↑leg cycle, no ↑PFT
Lake[41]	6	Arms	1 hour, 3 times/week	8	Several types	No change in P_{Imax}, VME
	6	Legs	1 hour, 3 times/week	8	Walking	No change in P_{Imax}, VME
	7	Arms & legs	1 hour, 3 times/week	8	Combined	No change in P_{Imax}, VME
Ries[42]	8	Gravity resistance, arms	15 minutes, once daily	6	Low resistance	↑arm endurance, high repetition, ↓dyspnea
	9	Neuromuscular facilitation	15 minutes, once daily	6	Weight lifts	↑arm endurance, ↓dyspnea
	11	Controls	—	6	Walk	No change
Epstein[13]	13	Arm	30 minutes, once daily	8	UAE	↑$\dot{V}_{O_2max}$ & $\dot{V}_E$, ↑P_{Imax}
	10	VMT	30 minutes, once daily	8	VMT	↑P_{Imax} & VME

*VMT = ventilatory muscle training; VME = ventilatory muscle endurance; PFT = pulmonary function tests; $\dot{V}_{O_2max}$ = peak oxygen uptake; $\dot{V}_E$ = minute ventilation; P_{Imax} = maximal inspiratory pressure; UAE = upper arm exercise.

RESPIRATORY MUSCLES & BREATHING TRAINING

It has been 18 years since Leith and Bradley[44] first demonstrated that, like their skeletal counterparts, the respiratory muscles of normal individuals can be specifically trained to improve their strength or their endurance. Subsequent to that observation, multiple studies have shown that a training response occurs if there is enough of a stimulus. An increase in inspiratory muscle strength (and perhaps endurance) should result in improved respiratory muscle function by decreasing the P_I/P_{Imax}, the ratio that represents the effort required to complete each breath (P_I), as a function of the force reserve (P_I/P_{Imax}). P_I/P_{Imax} has been shown to be the most important determinant for the development of fatigue in a loaded diaphragm.[45] Because reduced inspiratory muscle strength is evident in patients with COPD, efforts have been made to define the role of respiratory muscles training in these patients.

Ventilatory Muscle Strength & Endurance Training

Strength Training

To achieve increased strength, a high-intensity, low-frequency stimulus is needed. Inspiratory muscles are trained by performing inspiratory maneuvers against a closed glottis or shutter. Several studies have shown an increase in P_{Imax} when the respiratory muscles have been specifically trained for strength. Lecoq et al[46] studied 9 patients (some of whom had COPD) and after 4 weeks showed a 50% increase in P_{Imax}. Reid and Warren[47] observed a 53% increase in 6 COPD patients after 5 weeks of training. Both groups noticed a small but significant increase in expiratory muscle pressure. The clinical importance of strength training for the respiratory muscles has not been explored and, although decreasing P_I/P_{Imax} is theoretically important, it has not been shown to play a clinical role It is, nevertheless, important to point out that respiratory muscle strength has been shown to increase as a by-product of the endurance training achieved with the use of resistive loads. It is then possible that some of the observed benefits reported after endurance training may relate to the increased strength.

Endurance Training

Increased endurance is achieved by low-intensity, high-frequency training programs. The programs that have been used are of three types: flow-resistive loading, threshold loading, and voluntary isocapnic hyperpnea.

Flow-Resistive and Threshold Loading. In flow-resistive training, decreasing inspiratory breathing-hole size has determined load (ie, if breathing-hole size is decreased, load increases if frequency, tidal volume, and inspiratory time are held constant). Although most studies in patients with COPD have shown an improvement in the time that a given respiratory load can be maintained (VM endurance), the results have to be interpreted with caution because it has been shown that endurance can be influenced and actually increased with changes in the pattern of breathing. Threshold-loading has been employed and has been shown to result in some muscle training. This is done by assuring that the inspired pressure is at least high enough to ensure training, independent of inspiratory flowrate. Although breathing pattern is important (t_I and respiratory rate), it is not as critically important as is inspiratory pressure. Many studies have not been controlled and to interpret the results as a product of the training is difficult. The controlled studies summarized in Table 7 show an increase in the endurance time that the VMs can tolerate a known load;[48-56] some of them have shown a significant increase in strength[49-51] and a decrease in dyspnea to inspiratory load.[50] In the studies in which systemic exercise performance was evaluated, walking distance minimally increased.[48,49,56] It seems that the pressure required to achieve some training has to be at least 30% of maximal inspiratory pressure. In a recent study, Weiner and co-workers[57] randomized 36 COPD patients to 3 groups. Group 1 received specific VM threshold training combined with general exercise reconditioning. Group 2, received exercise training alone, whereas Group 3 received no training. VM training improved VM strength and endurance (as is already known), but patients treated with the combination of exercise and VM training manifested a significant increase in exercise tolerance when compared with those who only exercised. In a companion article,[58] the same group reported that asthmatic patients treated with VM

Table 7. Controlled Trials of Ventilatory Muscle Resistive Training in COPD

First Author	n	Type of Training	Frequency	Duration	Results
Pardy[48]	9	Resistive breathing	Twice daily	8 weeks	↑endurance time, ↑12-minute walk
	8	Physical therapy	3 times/week	8 weeks	No change
Larson[49]	10	Resistive breathing, 30% P_{Imax}	Once daily, 30 minutes	8 weeks	↑P_{Imax},* ↑endurance time, ↑12-minute walk
	12	Resistive breathing, 15% P_{Imax}	Once daily, 30 minutes	8 weeks	No ↑in P_{Imax}, endurance time, or 12-minute walk
Harver[50]	10	Resistive breathing	Twice daily, 15 minutes	8 weeks	↑P_{Imax}
	9	Sham	Twice daily, 15 minutes	8 weeks	No change
Belman[51]	8	Resistive breathing (high intensity)	Once daily, 30 minutes	6 weeks	↑endurance time, ↑P_{Imax} significantly higher in high intensity group vs low intensity group
	9	Resistive breathing (low intensity)	Once daily, 30 minutes	6 weeks	
Chen[52]	7	Resistive breathing	Once daily, 30 minutes	4 weeks	↑endurance time
	6	Sham	Once daily, 30 minutes	4 weeks	No change
Bjerre[53]	14	Resistive breathing	Once daily, 45 minutes	6 weeks	↑endurance time, no exercise
	14	Sham	Once daily, 45 minutes	6 weeks	No change
Falk[54]	12	Resistive breathing	Once daily, 45 minutes	12 months	↑endurance time, no exercise
	15	Sham	Once daily	12 months	No change
Noseda[55]	12	Resistive breathing	Once daily, 30 minutes	8 weeks	↑endurance time
	13	Breathing exercise	Once daily	8 weeks	No change
Jones[56]	7	Resistive breathing	Once daily, 30 minutes	10 weeks	↑exercise endurance
	6	Sham	Once daily	10 weeks	↑exercise endurance
	8	Exercise	Once daily	10 weeks	↑exercise endurance
Weiner[57]	12	Resistive breathing & exercise	Once daily	3 months	↑↑exercise ↑P_{Imax}
	12	Exercise	Once daily	3 months	↑ exercise
	12	Control	None	3 months	No change

*P_{Imax} = maximal inspiratory pressure.

training not only increased strength but also showed an improvement in asthma symptoms, hospitalizations, emergency department visits, school or work absenteeism, and medication consumption.

From the data obtained, it is clear that VM training with resistive breathing results in improved VM strength and endurance. In COPD, it is not clear whether this effort results in decreased morbidity or mortality or offers any clinical advantage that makes it worth the effort. In many of the studies, compliance (ie, adherence) was low, with up to 50% of patients failing to complete the studies. On the other hand, the study by Weiner and co-workers[58] suggests that this form of treatment for asthmatics should be further explored.

Ventilatory Isocapnic Hyperpnea. Ventilatory isocapnic hyperpnea is a training method by which patients maintain high levels of ventilation over time (15 minutes, 2 or 3 times daily). The oxygen and carbon dioxide concentrations in the breathing circuit are kept constant. The results of an uncontrolled study[59] show that after 6 weeks of training, the patients with COPD not only increased their maximal sustained ventilatory capacity but also increased arm- and leg-exercise performance. Two controlled studies (Table 8) also reported increases in maximal sustainable vital capacity in COPD in patients trained for 6 weeks, but the exercise endurance observed was no greater than that observed in the control group.[60,61]

Table 8. Controlled Trials of Ventilatory Isocapnic Hyperpnea in Patients with COPD

First Author	n	Type of Exercise	Frequency	Duration (weeks)	Results
Ries[60]	5	VIH*	45 min	6	↑MSVC ↑exercise
	7	Walking	45 min	6	↑exercise
Levine[61]	15	VIH	15 min	6	↑MSVC ↑exercise ↑ADL
	17	IPPB	15 min	6	↑ADL ↑exercise

*VIH = ventilatory isocapnic hyperpnea; MSVC = maximal sustainable ventilatory capacity; ADL = activities of daily living; and IPPB = intermittent positive pressure breathing.

It seems that respiratory muscle training results in increased strength and capacity of the muscles to endure a respiratory load; however, whether it also results in improved exercise performance or in improved performance of activities of daily living is still debated. From the respiratory muscle factors that may contribute to ventilatory limitation in COPD, it seems logical to predict that increases in strength and endurance should help respiratory muscle function, but this is perhaps only important in the capacity of the patients to handle inspiratory loads (for example, in acute exacerbations of their disease). It is less likely that VM training has a great impact on systemic exercise performance.

Ventilatory Muscle Training in the Patient in the ICU

Overview

Few objective data exist that allow a valid conclusion regarding the role of VM training in the ICU patient. It is apparent that as soon as a patient is left to breathe on his own (as during any form of weaning), his respiratory muscles are being retrained. Unconsciously, we have been using this methodology when we place patients on T-piece or low-rate synchronized mandatory intermittent ventilation (SIMV), but we have not analyzed results in terms of this being a training method. More often, we think of training in terms of the addition of an external load above and beyond spontaneous respiration. Little experience has been reported on patients who have recovered or are recovering from ventilatory failure. Belman reported improvement in two patients.[62] In a larger but still uncontrolled study, Aldrich et al[63] recruited 30 patients with stable chronic respiratory failure for at least 3 weeks who had failed repeated weaning attempts. Patients with active infections or unstable cardiovascular, renal, or endocrine problems were not included. The authors also excluded patients with gross malnutrition (albumin < 2.5 g/dL) and/or neuromuscular disease. The patients were intermittently trained by having them breath through an inspiratory resistor while they breathed spontaneously or were supported by SIMV 2 to 8 breaths/minute. In these patients, P_{Imax} improved from mean (SD) –37 (15) cm H_2O to –46 (15) cm H_2O, whereas vital capacity increased from 561 (325) mL to 901 (480) mL. Of the 30 patients, 12 were weaned after 46 days of training (40% success). Because the study was uncontrolled and used a selected group of patients, these findings may not apply to most patients recovering from respiratory failure, and the success rate is not much different from those reported in weaning facilities that have not used VM training.[64] In spite of these encouraging reports, before VM training can be recommended as a form of treatment for patients with respiratory failure, more rigorous studies need to be completed.

Finally, it is important to state that VM training, especially with resistive- or threshold-loading, may be deleterious. It has been shown that breathing at high P_I/P_{Imax} or prolonged inspiratory time/respiratory cycle time, or t_I/t_{tot}, may induce muscle fatigue.[65] Both factors are an intrinsic part of this form of training, and, thus, enforcing a program intense enough may actually precipitate fatigue.

Breathing Retraining

Other less conventional forms of training exist that are open to critical review, are conceptually solid, and may offer new avenues of treatment. As we have seen, the ventilated patient has a high ventilatory drive. As a matter of fact, it has been shown that patients who fail a ventilator weaning trial, manifest higher drive than those patients who wean successfully. I review the limited data available.

Feedback. In 1990, Holliday and Hyers[66] studied 40 patients after at least 7 days of mechanical ventilation. Patients were randomized to conventional weaning or weaning with electromyographic feedback using the frontalis signal as an indicator of tension and to induce relaxation. They also used surface EMG of intercostals and diaphragm as indicators of respiratory muscle activity. Use of feedback signals to encourage relaxation and larger tidal volumes, produced differences between treated and untreated patients.

The results indicate a reduction in mean ventilator days for the biofeedback group. Tidal volume (V_T) and tidal volume-inspiratory time ratio (V_T/t_I) increased significantly for this group. The increase was also significant when corrected by diaphragmatic-EMG amplitude, which was interpreted as improved diaphragmatic efficiency. The authors[66] concluded that breathing retraining resulted in a more efficient breathing pattern, which in turn decreased dyspnea and anxiety and allowed for quicker weaning time in the treated patients. My group[67] has studied some of these factors. Work of breathing as determined by the pressure-time integral of the excursions of the continuously recorded P_{pl} ($f\int P_{es}dt$) was measured before and after rehabilitation in 16 patients with COPD. In these patients, pulmonary functions did not change but $f\int P_{es}dt$ at the exercise isotime (defined as the same time during both forms of exercise) after rehabilitation decreased significantly (Table 9). This drop was mostly due to a decrease in respiratory frequency.[67] Finally, retraining in breathing techniques that decrease breathing frequency (or pursed-lip breathing, PLB) has been shown to result in increases in V_T and oxygen saturation and decreases in dyspnea.[68]

Table 9. Work of Breathing, Exercise Endurance, and Maximal Transdiaphragmatic Pressure before and after Pulmonary Rehabilitation

Rehabilitation	Endurance time (s)	$f\int P_{es}dt$* (cm $H_2O \cdot min^{-1}$)	P_{dimax} (cm H_2O)
Before	434	288	48
After	512†	219†	52

*$f\int P_{es}dt$ = work of breathing as estimated by the pressure-time index calculated from continuous recording of esophageal pressure (P_{pl}); P_{dimax} = maximal transdiaphragmatic pressure.
†$p < 0.05$.

In the work by Epstein et al from our laboratory,[13] of the many factors that may have contributed to improved exercise endurance for upper extremity exercise after upper extremity training, the most striking was a drop in V_T/t_I at exercise isotime. We believe this may represent better coordination of the respiratory muscles.

Yoga. Yoga is a philosophical doctrine that includes control of posture and voluntary control of breathing. The latter includes slow, deep breaths with apnea at end of inspiration and expiration and/or utilization of rapid abdominal maneuvers. The breathing rate may be brought down to 4-6 breaths/minute. Stanescu et al[69] compared the breathing pattern of 8 well-trained yoga practitioners with 8 sex-, age-, and height-matched controls. The yoga group had a pattern of breathing characterized by ample V_T and slow breathing frequency. They also had a lower ventilatory response to CO_2 rebreathing. The mechanism by which this occurs is not clear but may be habituation to chronic overstimulation of stretch receptors. Again, it is possible that because ventilation is automatically controlled by structures in the upper medulla and brain stem and voluntarily by the cortex, sustained slow, deep breathing may become a 'learned' reflex. Whatever the mechanism, this may have applications. Tandon[70] studied patients with chronic airway obstruction trained in yoga breathing and compared them with controls. The trained patients controlled dyspnea better and improved their exercise tolerance.

Postural Changes. It is known that musculoskeletal tone and contraction may be determined by habitual positioning. Over the last few years, increasing attention has been given to the voluntary inhibition of those patterns. Recently, Austin and Pullin[71] demonstrated improved peak expiratory flowrate, maximal voluntary ventilation, and maximal inspiratory and expiratory pressures in normal subjects who underwent lessons in proprioceptive musculoskeletal education compared to controls. The effects of proprioceptive musculoskeletal education per se have not been systematically evaluated in patients with lung disease but breathing retraining (PLB and diaphragmatic breathing) constitutes a form of therapy that resembles the techniques.

Pursed-Lip Breathing. Indeed, PLB results in slowing of the breathing rate with increases in V_T. As Roa et al showed in our laboratory,[68] PLB results in a shift in the pattern of recruitment of the VMs from one that is predominantly diaphragmatic to one that recruits more of the accessory muscles of the rib cage and the abdominal muscles of exhalation. Perhaps this shift may contribute to the relief of dyspnea that has been reported by patients when the technique is adopted. Patients on ventilators cannot purse-lip breathe but in some ventilator patients respiratory retard or PEEP does improve oxygenation, decrease respiratory rate, augment ventilation, and decrease work of breathing in weaning patients. PLB and PEEP may have similar physiologic effects and may make the former therapy indicated once the latter has been discontinued.

IN SUMMARY

In summary, there are more unresolved questions than absolute facts related to training and respiratory muscle function. A wealth of information remains to be gained if systematic scientific analysis is applied to answer the questions I have addressed in this review. It is rewarding to see that widespread interest in applied respiratory physiology has begun to produce results that may benefit the large number of patients suffering from disabling respiratory diseases and for whom no other viable therapeutic options exist. There is every reason to believe that all of us involved in direct patient care may, in the future, be able to apply relatively simple programs to enhance quality of life.

REFERENCES

1. Clausen JP, Klausen K, Rasmussen B, Trap-Jensen J. Central and peripheral circulatory changes after training of the arms or legs. Am J Physiol 1973;225(3):675-682.
2. Davies CT, Sargeant AJ. Effects of training on the physiological responses to one- and two-leg work. J Appl Physiol 1975;38(3):377-385.
3. Belman MJ, Kendregan BA. Exercise training fails to increase skeletal muscle enzymes in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1981;123(3):256-261.
4. Siegel W, Blomqvist G, Mitchell JH. Effects of a quantitated physical training program on middle-aged sedentary men. Circulation 1970;41(1):19-29.
5. Niederman MS, Clemente PH, Fein AM, Feinsilver SH, Robinson DA, Ilowite JS, et al. Benefits of a multidisciplinary pulmonary rehabilitation program: improvements are independent of lung function. Chest 1991; 99(4):798-804.
6. Mohsenifar Z, Horak D, Brown HV, Koerner SK. Sensitive indices of improvement in a pulmonary rehabilitation program. Chest 1983;83(2):189-192.
7. Holle RH, Williams DV, Vandree JC, Starks GL, Schoene RB. Increased muscle efficiency and sustained benefits in an outpatient community hospital-based pulmonary rehabilitation program. Chest 1988;94(6):1161-1168.
8. Zack MB, Palange AV. Oxygen supplemented exercise of ventilatory and nonventilatory muscles in pulmonary rehabilitation. Chest 1985;88(5):669-675.
9. ZuWallack RL, Patel K, Reardon JZ, Clark BA 3rd, Normandin EA. Predictors of improvement in the 12-minute walking distance following a six-week outpatient pulmonary rehabilitation program. Chest 1991;99(4): 805-808.
10. Casaburi R, Patessio A, Ioli F, Zanaboni S, Donner CF, Wasserman K. Reductions in exercise lactic acidosis and ventilation as a result of exercise training in patients with obstructive lung disease. Am Rev Respir Dis 1991; 143(1):9-18.
11. Make BJ, Buchholz J. Exercise training in COPD patients improves cardiac function (abstract). Am Rev Respir Dis 1991;143(4, Part 2):80A.
12. O'Donnell DE, Webb KA, McGuire MA. Older patients with COPD: benefits of exercise training. Geriatrics 1993;48(1):59-62,65-66.

13. Epstein S, Breslin E, Roa J, Celli B. Impact of unsupported arm training (AT) and ventilatory muscle training (VMT) on the metabolic and ventilatory consequences of unsupported arm elevation (UAE) and exercise (UAEx) in patients with chronic airflow obstruction (CAO) (abstract). Am Rev Respir Dis 1991;143(4, Part 2):81A.
14. Saltin B, Blomqvist G, Mitchell JH, Johnson RL Jr, Wildenthal K, Chapman CB. Response to exercise after bed rest and after training. Circulation 1968;38(5, Suppl):1-78.
15. Keens TG, Krastins IR, Wannamaker EM, Levison H, Crozier DN, Bryan AC. Ventilatory muscle endurance training in normal subjects and patients with cystic fibrosis. Am Rev Respir Dis 1977;116(5):853-860.
16. Moser KM, Bokinsky GE, Savage RT, Archibald CT, Hansen PR. Results of a comprehensive rehabilitation program: physiologic and functional effects on patients with chronic obstructive pulmonary disease. Arch Intern Med 1980;140(12):1596-1601.
17. Beaumont A, Cockcroft A, Guz A. A self-paced treadmill walking test for breathless patients. Thorax 1985; 40(6):459-464.
18. Christie D. Physical training in chronic obstructive lung disease. Br Med J 1968;2(598):150-151.
19. Hughes RL, Davison R. Limitations of exercise reconditioning in COLD. Chest 1983;83(2):241-249.
20. Paez PN, Phillipson EA, Masangkay M, Sproule BJ. The physiologic basis of training patients with emphysema. Am Rev Respir Dis 1967;95(6):944-953.
21. Cockcroft AE, Saunders MJ, Berry G. Randomized controlled trial of rehabilitation in chronic respiratory disability. Thorax 1981;36:200-203.
22. Sinclair DJ, Ingram CG. Controlled trial of supervised exercise training in chronic bronchitis. Br Med J 1980;280(6213):519-521.
23. Woolf CR, Suero JT. Alterations in lung mechanics and gas exchange following training in chronic obstructive lung disease. Dis Chest 1969;55(1):37-44.
24. McGavin CR, Gupta SP, McHardy GJ. Twelve-minute walking test for assessing disability in chronic bronchitis. Br Med J 1976;1(6013):822-823.
25. Pollock M, Roa J, Benditt J, Celli B. Estimation of ventilatory reserve by stair climbing: a study in patients with chronic airflow obstruction. Chest 1993;104(5):1378-1383.
26. O'Hara WJ, Lasachuk KE, Matheson PC, Renahan MC, Schlotter DG, Lilker ES. Weight training and backpacking in chronic obstructive pulmonary disease. Respir Care 1984;29(12):1202-1210.
27. Martinez FJ, Couser JI, Celli BR. Factors influencing ventilatory muscle recruitment in patients with chronic airflow obstruction. Am Rev Respir Dis 1990;142(2): 276-282.
28. Tangri S, Woolf CR. The breathing pattern in chronic obstructive lung disease during the performance of some common daily activities. Chest 1973;63(1):126-127.
29. Celli BR, Rassulo J, Make BJ. Dyssynchronous breathing during arm but not leg exercise in patients with chronic airflow obstruction. N Engl J Med 1986;314 (23):1485-1490.
30. Criner GJ, Celli BR. Effect of unsupported arm exercise on ventilatory muscle recruitment in patients with severe chronic airflow obstruction. Am Rev Respir Dis 1988; 138(4):856-861.
31. Celli B, Criner G, Rassulo J. Ventilatory muscle recruitment during unsupported arm exercise in normal subjects. J Appl Physiol 1988;64(5):1936-1941.
32. Bobbert AC. Physiological comparison of three types of ergometry. J Appl Physiol 1960;15:1007-1014.
33. Davis JA, Vodak P, Wilmore JH, Vodak J, Kurtz P. Anaerobic threshold and maximal aerobic power for three modes of exercise. J Appl Physiol 1976;41(4):544-550.
34. Stenberg J, Astrand PO, Ekblom B, Royce J, Saltin P. Hemodynamic response to work with different muscle groups, sitting and supine. J Appl Physiol 1967;22(1): 61-70.
35. Reybrouck T, Heigenhauser GF, Faulkner JA. Limitations to maximum oxygen uptake in arm, leg and combined arm-leg ergometry. J Appl Physiol 1975;38(5): 774-779.
36. Martin TW, Zeballos RJ, Weisman IM. Gas exchange during maximal upper extremity exercise. Chest 1991; 99(2):420-425.
37. Banzett RB, Topulos GP, Leith DE, Nations CS. Bracing arms increases the capacity for sustained hyperpnea. Am Rev Respir Dis 1988;138(1):106-109.
38. Maestro L, Dolmage T, Avendano MA, Goldstein R. Influence of arm position in ventilation during incremental exercise in healthy individuals (abstract). Chest 1990; 98(2, Suppl):113s.
39. Dolmage TE, Maestro L, Avendano M, Goldstein RS. The ventilatory response to arm elevation of patients with chronic obstructive pulmonary disease. Chest 1993; 104:1097-1100.
40. Couser JI Jr, Martinez FJ, Celli BR. Respiratory response and ventilatory muscle recruitment during arm elevation in normal subjects. Chest 1992;101(2):336-340.
41. Lake FR, Henderson K, Briffa T, Openshaw J, Musk AW. Upper-limb and lower-limb exercise training in patients with chronic airflow obstruction. Chest 1990; 97(5):1077-1082.
42. Ries AL, Ellis B, Hawkins RW. Upper extremity exercise training in chronic obstructive pulmonary disease. Chest 1988;93(4):688-692.
43. Couser J, Martinez F, Celli B. Effect of pulmonary rehabilitation (PR) including arm exercise on ventilatory muscle function during arm elevation (AE) in patients with chronic airflow obstruction (CAO) (abstract). Am Rev Respir Dis 1989;139(4, Part 2):232A.
44. Leith DE, Bradley M. Ventilatory muscle strength and endurance training. J Appl Physiol 1976;4:508-516.
45. Roussos C, Macklem PT. The respiratory muscles. N Engl J Med 1982;307(13):786-797.
46. Lecoq A, Delhez L, Janssens S, Petit JM. Reentrainement de la fonction motrice ventilatoires chez des insuff-

isants respiratoire chroniques. Acta Tuberc Pneumol Belg 1970;61(1):63-69.

47. Reid WD, Warren CP. Ventilatory muscle strength and endurance training in elderly subjects and patients with chronic airflow limitation: a pilot study. Physiol Canada 1984;36:305-311.
48. Pardy RL, Rivington RN, Despas PJ, Macklem PT. Inspiratory muscle training compared with physiotherapy in patients with chronic airflow limitation. Am Rev Respir Dis 1981;123(4, Part 1):421-425.
49. Larson JL, Kim MJ, Sharp JT, Larson DA. Inspiratory muscle training with a pressure threshold breathing device in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1988;138(3):689-696.
50. Harver A, Mahler DA, Daubenspeck JA. Targeted inspiratory muscle training improves respiratory muscle function and reduces dyspnea in patients with chronic obstructive pulmonary disease. Ann Intern Med 1989; 111(2):117-124.
51. Belman MJ, Shadmehr R. Targeted resistive ventilatory muscle training in chronic obstructive pulmonary disease. J Appl Physiol 1988;65(6):2726-2735.
52. Chen H, Dukes R, Martin BJ. Inspiratory muscle training in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1985;131(2):251-255.
53. Bjerre-Jepsen K, Secher NH, Kok-Jensen A. Inspiratory resistance training in severe chronic obstructive pulmonary disease. Eur J Respir Dis 1981;62(6):405-411.
54. Falk P, Eriksen AM, Kolliker K, Andersen JB. Relieving dyspnea with an inexpensive and simple method in patients with severe chronic airflow limitation. Eur J Respir Dis 1985;66(3):181-186.
55. Noseda A, Carpiaux JP, Vandeput W, Prigogine T, Schmerber J. Resistive inspiratory muscle training and exercise performance in COPD patients: a comparative study with conventional breathing retraining. Bull Eur Physiopathol Respir 1987;23(5):457-463.
56. Jones DT, Thomson RJ, Sears MR. Physical exercise and resistive breathing training in severe chronic airways obstruction—are they effective? Eur J Respir Dis 1985; 67(3):159-166.
57. Weiner P, Azgad Y, Ganam R. Inspiratory muscle training, combined with general exercise reconditioning in patients with COPD. Chest 1992;102(5):1351-1356.
58. Weiner P, Azgad Y, Ganam R, Weiner M. Inspiratory muscle training in patients with bronchial asthma. Chest 1992;102(5):1357-1361.
59. Belman MJ, Mittman C. Ventilatory muscle training improves exercise capacity in chronic obstructive pulmonary disease patients. Am Rev Respir Dis 1980; 121(2):273-280.
60. Ries AL, Moser KM. Comparison of isocapnic hyperventilation and walking exercise training at home in pulmonary rehabilitation. Chest 1986;90(2):285-289.
61. Levine S, Weiser P, Gillen J. Evaluation of a ventilatory muscle endurance training program in the rehabilitation of patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1986;133(3):400-406.
62. Belman MJ. Respiratory failure treated by ventilatory muscle training (VMT): a report of two cases. Eur J Respir Dis 1981;62(6):391-395.
63. Aldrich TK, Karpel JP, Uhrlass RM, Sparapani MA, Eramo D, Ferranti R. Weaning from mechanical ventilation: adjunctive use of inspiratory muscle resistive training. Crit Care Med 1989;17(2):143-147.
64. Scheinholm DJ, Ho C. Avoiding home mechanical ventilation; the regional weaning center (abstract). Proceedings of the International Conference on Pulmonary Rehabilitation and Home Ventilation held in Denver, Colorado 1991;3:A35.
65. Bellemare F, Grassino A. Evaluation of human diaphragm fatigue. J Appl Physiol 1982;53(5):1196-1206.
66. Holliday JE, Hyers TM. The reduction of weaning time from mechanical ventilation using tidal volume and relaxation biofeedback. Am Rev Respir Dis 1990;141(5, Part 1):1214-1220.
67. Benditt J, Pollock M, Breslin E, Celli B. Comprehensive pulmonary rehabilitation decreases the work of breathing during leg cycle ergometry in patients with severe chronic airflow obstruction (CAO) (abstract). Am Rev Respir Dis 1990;141(4, Part 2):A509.
68. Roa J, Epstein S, Breslin E, Shannon T, Celli B. Work of breathing and ventilatory muscle recruitment during pursed lip breathing in patients with chronic airway obstruction (abstract). Am Rev Respir Dis 1991;143(4, Part 2):A77.
69. Stanescu DC, Nemery B, Veriter C, Marechal C. Pattern of breathing and ventilatory response to CO_2 in subjects practicing hatha-yoga. J Appl Physiol 1981;51(6):1625-1629.
70. Tandon M. Adjunct treatment with yoga in chronic severe airways obstruction. Thorax 1978;33(4):514-517.
71. Austin J, Pullin G. Improved respiratory function after lessons in the Alexander technique of musculoskeletal education (abstract). Am Rev Respir Dis 1984;129(4, Part 2):A275.

Discussion begins on next page.

Celli Discussion

Fletcher: I have a question about outcome. Do you agree, and will you also give me your figures—of the people who exercise and improve (improve their feeling of well-being and exercise capacity), how many don't maintain the exercise, and lose the improvement.

Celli: I agree and let me tell you my personal experience, then I'll give you some data. Yes, they do better. Yes, they stop doing it—8 out of 10, I'm afraid, stop doing it. The reason why I say this is that at our VA (we don't have a maintenance program because we don't have the means to do so) about 20-30% of our patients are reruns. They are the patients who come back after 8 or 9 months and say, "Dr Celli, I'd like to do it again because I felt better." That's my personal experience, and I think it's the experience of most people who do this. There is a group in Connecticut headed by Richard ZuWallack and Jane Reardon. They have a large stadium that they can use. They do maintenance once a week or so, and are able to provide us with data that show that if you structure patients into a club of better breathers, where reinforcement is given by each individual, and you put them there once a week, they tend to maintain the benefit. I don't know his data; he hasn't published it in terms of how many of those actually drop out with time, so I cannot answer that, unfortunately. But I would say that 20% of people will continue to do it.

Pierson: Ordinary people without lung disease know that if they exercise, it's good for them. Yet only a relatively small proportion of the population does this. A lot of people who take up exercise programs find that it doesn't fit in with their lives very well, and they tend to fall away from them. In practical terms, how do you combat this in your patient population? The natural tendency for many people is not to exercise. How do you get them to do this?

Celli: Well, unfortunately, if you look at any rehab criteria, the first thing is that the patient has to be willing to participate. So, what you're asking me right off the bat is, How many have we skimmed and not booted? I would say that the majority will never go into a rehab program because they are the patients who perhaps would not exercise anyway. I'd rather sit down by the pool than exercise; so, if I had COPD and were asked to join a rehab program, I would probably be less prone to do so than to change my old habits.

Pierson: You may be more motivated by virtue of your COPD because it shows you how disabled you are, I suppose.

Celli: There are no data that I can tell you. The best trick is the patient talking to other people. And I'll give you, again, an anecdote. I have a patient who lives in Brighton, Massachusetts, who trained with us at the VA. He kept on coming, and what he did was to set up a program for the Mattapan area where he lives. He lives in a poor section, and most of his friends do not have access to health insurance. He's running his own rehab program in Mattapan, for his buddies who smoked, who are doing the exercises with him. So, in that spectrum, sometimes you get this jewel who will do things that will surprise you. For the last 7 years he's been doing this in Mattapan with a bunch of other guys who do not have insurance and are not dead.

Sortor-Leger: I think the other point—I don't remember whether you made it or not, is intervention during an acute episode. And that's how we get these patients to stop smoking. It's also, Dave (Pierson), how we can get them interested in wellness and in something like this, is by saying "You're really sick but if you quit smoking and start exercising you'll feel better."

Celli: I agree with that, and I would like to tell you that in order to get into our rehab program, they invariably are asked to swallow balloons, before and after, and our compliance rate is about 90%. They must feel better, if they're willing to come to me and allow me to put balloons down their esophagus and count to 12. So, I believe that patients do feel better.

Bach: I want to ask you about the patient who apparently died despite the inspiratory muscle training. Was he severely hypercapnic?

Celli: No, he was not. He didn't die. He had two ventilatory failure episodes and he recovered.

Hardy: Is anybody looking at shorter durations of exercise that could be better incorporated into daily life? Healthy people obviously also have problems motivating themselves to exercise per the old adage of 30 minutes 3 times a week. Newspapers and popular media are now coming out saying all you need to do is quit using the elevator and use the stairs for short bursts throughout the day, and you will become more fit. Is anybody starting to look at this population, for example, with shorter bursts? You had a 30-minute regimen for your study—that's a long time. Have you started to look at shorter durations of some kind of work that is 'normal,' as it were? Something like climbing stairs that can be incorporated into their lives?

Celli: We haven't started looking at that because we're very interested in physiology, more than we have been interested in other outcomes. Although I have developed interest in the patient's outcome, we have been following principles of training that have been proven over time. But I can tell you this, that Michael Bellman has a very nice talk on exercise, and he's compared what he calls "laissez faire" exercise programs with "structured" exercise programs and shown that it doesn't make a big difference. The "laissez faire" patients may have as good an outcome. So your question is very well taken, but I cannot give you my own personal experience on how short it could be.

Make: I think the comments about maintenance are very important. Guyatt's study indicates that the benefits of pulmonary rehabilitation in terms of exercise and quality of life decrease over a period of a year.[1] Twenty-four of 31 patients benefitted from pulmonary rehabilitation over a period of 6 weeks; however, only 11 of the 24 maintained their gains at 6 months. On the other hand, our preliminary data show that quality of life gains are maintained for a year after rehabilitation. It may be that in a chronic disease that has a progressive downhill course, maintenance of quality of life may be an improvement compared to controls who have a decline in quality of life. There's very little in the literature about how to provide a maintenance program for these patients. Most investigators have been interested in the acute effects of rehabilitation, and patients may see rehabilitation as a short-term and not life-long treatment program. It's very difficult to get patients to continue to exercise in the community. Even though we give our patients lots of different ideas on how and where to continue to exercise and make follow-up phone calls to assess adherence, it's still difficult for them to incorporate exercise into their daily lives. If we think about how we might continue an exercise program, you and I might join a health club, YMCA, or community recreation center. These patients don't like going to these settings because they are often embarrassed by their illness and upset when they compare themselves to others without respiratory disease. Bart's idea of forming a group where these patients can congregate has merit. That's best done in the community, and unfortunately many rehabilitation programs are not community based.

REFERENCE

1. Guyatt GN, Berman LB, Townsend M. Long-term outcome after respiratory rehabilitation. Can Med Assoc J 1987;137(12):1089-1095.

Celli: I do believe that ZuWallack's idea is excellent. I believe he obtained permission to use a little stadium at the University of Connecticut, and he meets there once a week with this huge group—like 50—of COPDers sitting there. They walk around the track; they have a good time. They talk about everything, and they seem to do pretty well. So, that maintenance issue will become an area to be studied more objectively.

Dunne: Bart, I was intrigued with the notion of using the backpack to increase exercise tolerance.[1,2] Have you ever heard of anyone in pulmonary rehabilitation using ankle weights like those used by basketball players? You know, adding a little poundage to stress the walk a bit more?

REFERENCES

1. O'Hara WJ, Lasachuk KE, Matheson PC, Renahan MC, Schlotter DG, Lilker ES. Weight training and backpacking in chronic obstructive pulmonary disease. Respir Care 1984;29(12):1202-1210.
2. O'Hara WJ, Lasachuk KE, Matheson PC, Renahan MC, Schlotter DG, Lilker ES. Weight training benefits in chronic obstructive pulmonary disease: a crossover controlled study. Respir Care 1987;32(8):660-668.

Celli: No, I have not heard of people systematically doing that. Somebody made the comment about walking up stairs. It is very interesting. We're using bikes or electrically braked ergometers. We have a study coming out in *Chest*[1] in which we took 45 COPDers, and we walked them up stairs. We measured oxygen uptake at each flight. We had a giant mouthpiece with 6 flanges, and we took 6 Douglas bags and 4 Fellows and (to make a long story short) we were able to measure oxygen uptake at each landing. It was a lot of work, but it was worth it because what we did is we compared those $\dot{V}_{O_2}$s and $\dot{V}_{CO_2}$s at each level with an exercise study on a bicycle ergometer and showed that the predictability of oxygen uptake walking upstairs was as good as the relationship that exists between mechanical work load and oxygen uptake. So, you could, on those patients on whom the 6-minute walk may not be high enough to reach their limit, complete a stair climbing protocol. I think we underestimate the possibility of using stairs. Let me just add that the reviewer botched it the first time. He said, "Oh, you don't know how high is the step, how deep is the step, how high the flights"; so I had to go around Boston and measure the steps and the flights of 6 different hospitals. I found that the height of steps at the VA and a community hospital are fairly consistent. The mean height for a hospital floor is 3.3 ± 0.1 meter. And the mean height of a step is 17 cm ± 0.1. So it's a very narrow height spread. I believe that if we simply use steps and stairs, we can achieve excellent results.

REFERENCE

1. Pollock M, Roa J, Benditt J, Celli B. Estimation of ventilatory reserve by stair climbing: a study in patients with chronic air flow obstruction. Chest 1993;104(5):1378-1383.

Hill: Bart, one of your concluding remarks was related to breathing exercises, and I think you showed some pretty convincing data. We all know that there are data that say pursed-lip breathing is worth teaching. What other breathing exercises are worth teaching patients?

Celli: You know, there is very little on that. I can tell you my bias. I think the crucial question, the physiologically crucial question, is whether the respiratory centers can only respond to stimuli in one way. Nobody has addressed that. That is to say, if I give you CO_2, you'll increase the depth and the rate of your breathing. If I put you on a bike, you'll increase the depth and the rate of your breathing. If I ask you to breathe, you increase the depth and the rate of your breathing. Perhaps we have like a visceral thing in there that cannot be modified. That is one scenario—in which breathing retraining is only good while the guy consciously thinks about it. The other scenario is that perhaps it is not like that. Perhaps the neurons in there can interact in such a way that the cortex could be retrained to do this better, and there is some evidence for that. Somebody asked me if I had ever been to India. John (Bach), you asked me that. I have not, but I have read about yoga. If you take individuals and you train them in yoga, and then you give them a CO_2 rebreathing curve before and after yoga training, their slopes drop significantly. So, now their respiratory centers appear to respond to a rise in CO_2 that is not as steep as before yoga training. Based on that, there are a couple of papers where normal individuals were taught to breathe slowly and deeply. They hold their breath for longer periods, and have gotten rates to as low as 6 per minute,[1,2] and I believe there is a whole world in there that remains to be explored. The ATS has a session next May just on controlled breathing that will be looking at all the different aspects. I believe that we've underestimated that.

REFERENCES

1. Stanescu D, Nemery B, Veriter C, Marechal C. Pattern of breathing and ventilatory response to CO_2 in subjects practicing hatha-yoga. J Appl Physiol 1981;51(6):1625-1629.
2. Tandon MK. Adjunct treatment with yoga in chronic severe airways obstruction. Thorax 1978;33(4):514-517.

Gilmartin: I have a couple of comments. First, I thought in Boston we used beer cans for the patients' weights.

Celli: That was only for the Irish, Mary.

Gilmartin: Well, they're all Irish. I think controlled breathing during exercise is really important to patients. The COPD patients that I worked with a lot when I was at the VA (or when I was at Beth Israel in Boston) were taught controlled diaphragmatic, relaxed breathing exercises during activities. I can remember patients who would walk up three or four flights of stairs, and the physician would say, "No, they couldn't be doing that. Their pulmonary function testing must be wrong." They would repeat pulmonary function testing. They were just as severe then as they had been in their previous testing, but they could walk up three or four flights of stairs with controlled breathing exercises.

Hill: Using diaphragmatic breathing?

Gilmartin: Using diaphragmatic breathing. But it takes a lot of work with those patients, because obviously you have to be with them, and it's a whole retraining and relearning thing. Because most patients with COPD tend to do things very quickly—all their lives they have done things quickly. Now you're telling them to slow down and concentrate on breathing. So it's very difficult, but it really works. The other comment I wanted to make about exercise is that I think we need to be careful when we exercise patients in a controlled environment—either by bike exercise or even upper extremity exercise—that we have something that carries over to what they're going to do at home. Because patients on a bike may do well, they don't desaturate and they don't get out of breath because I think they have fixed their upper extremity muscles by holding onto the handlebars. Then you get them walking or you get them doing just their normal activities, they desaturate and they get much more short of breath. In our rehabilitation program, we may train the patient on a bike, but we also look at what they are going to be doing at home and what they can do. If they can't ride a bike when they're at home, then you have to find what exercise they can do. Or, if they can't walk because they live in a bad area where they could get mugged or whatever, then you have to organize some other type of program. Whatever program they will be doing at home, the therapists must work with the patients on this type of exercise.

Bach: Just to mention it—you talk about breathing retraining, at least for the neuromuscular patient (who we really haven't considered very much here), you can see many who are very advanced with very low vital capacities, who are severely hypercapnic, often receiving oxygen therapy, and whose baseline saturations on room air are in the 40s to 70s when they're asleep and in the 80s when they're awake. These patients breathe rapidly and shallowly. You can retrain their breathing and reset their chemotactic centers by using oximetry biofeedback. The patient is instructed to take deep enough breaths to normalize oxyhemoglobin saturation without oxygen therapy. When he tires or desaturates, he is instructed to take deep breaths from a ventilator through a nosepiece or a mouthpiece. Although for a time, until compensatory metabolic alkalosis is reversed, the patient may need to use his ventilator up to 24 hours a day, with time he becomes able to maintain normal saturations when breathing on his own for increasingly longer periods of time. So, in a way, he's been doing breathing retraining by using oximeter biofeedback.

Celli: I didn't show you the slide of Holliday's work,[1] but he measured tidal volume and electromyographic activity of the diaphragm and showed that the relationship of electromyographic activity of the diaphragm to tidal volume dropped. That is to say, he took deeper breaths and stimulated the diaphragm to equal electric intensity, indicating more efficiency. That would support what you've just said.

REFERENCE

1. Holliday J, Hyers TM. The reduction of weaning time from mechanical ventilation using tidal volume and relaxation biofeedback. Am Rev Respir Dis 1990;141(5, Part 1):1214-1220.

Bach: You are also resting the muscles while you're ventilating the lung. That increases the patient's endurance and strength when he is breathing on his own.

Make: I have two observations about breathing patterns. First, probably the major benefit for controlled breathing (pursed-lip breathing, diaphragmatic breathing, or controlled breathing) is to allow patients to control their dyspnea. If patients adopt these techniques when they are short of breath, they became less short of breath. Controlled breathing can thus be considered as one part of collaborative self-management.

Hardy: Ellen Breslin has studied dyspnea and pursed-lip breathing.

Celli: Yes, but dyspnea did not change. Remember, they were resting. The data on dyspnea that changes with pursed-lip breathing are on exercise. But, again, that makes sense to me. I'll tell you why. Breslin did it in our lab, and the visual analog scale showed a value about 2.5 cm for resting COPDers. With pursed-lip breathing it went down to 1.9. One thing that amazes me is that I take my Fellows and I ask them and they'll point out 0.5 cm on the dyspnea scale at rest. You take a COPDer, he's 1.2. He's not 2 . . ., but you look at my Fellow, they look pretty good. You look at a COPDer and he looks like he's dying. If I were to rate his dyspnea, I'd mark it at 9. But he says, "Oh, reckon it at 1." When you put him on the bike, the COPDer doubling his ventilation goes to 8.2 cm of dyspnea. The intervention will indeed drop the visual analog scale to a significantly lower level if you purse-lip breathe on the exercise. So, when they're at rest, it doesn't make a big difference because they're not very afflicted, which goes back to Barry's (Make) point that if they were walking, exercising, or having acute exacerbation, then pursed-lip breathing could be very good.

Make: The other comment I have is that if you have ever gone to exercise at a health club, what's the first thing that the trainer tells you? He says, "Don't hold your breath." Most of us tend to breath-hold during an intense effort. If you watch patients with COPD, they're not unlike other people who are performing strenuous activity. It's just that for the patients, walking is more strenuous than it is for us. During a strenuous activity, patients have a tendency to alter their breathing pattern and develop irregular breathing, which may lead to decreases in oxygen saturation and increased dyspnea. Controlled breathing with activity is an important technique that has not been very well studied. We need to make sure that patients continue to breathe with a regular rhythmic pattern during an activity, and they need to know when to breathe. Do they breathe when going up the step or at the top of the step?

Sortor-Leger: I just want to raise a little bit of a flag in talking about the neuromuscular patient and breathing exercises and breathing control because I think we're mixing apples and oranges in that discussion. When we're working with neuromuscular patients who are retaining CO_2 and underventilating and probably fatigued, and we talk about exercising or working with breathing control, I think we're really looking in the wrong area. If we look at these patients and say that breathing control is effecting their better performance, I think there's absolutely no literature and a real big question in regard to that. I think it's dangerous to exercise these patients, and I think it's really dangerous to mix discussion about respiratory muscle exercise and breathing control in neuromuscular patients and the COPD patients. It's a different group of patients and the mechanisms are really different.

Bach: Well, first of all, even for the COPD patient, Foster's study in '88[1] showed that even severe hypercapnia doesn't preclude the COPD patient from benefitting from therapeutic exercise. Second, hypercapnia does not mean that the patient's respiratory muscles are fatigued. That's been brought out many times. What it is an indication of is that the patient is taking shallow breaths to prevent fatigue, and that's why the CO_2 goes up. If you look at the diaphragm, electromyographically, you're going to find that the diaphragm is not fatigued.

REFERENCE

1. Foster S, Lopez D, Thomas HM 3rd. Pulmonary rehabilitation in COPD patients with elevated P_{CO_2}. Am Rev Respir Dis 1988;138(6):1519-1523.

Sortor-Leger: You're talking about COPD, John?

Bach: In anybody.

Sortor-Leger: Quote me some literature on the neuromuscular patient.

Bach: There's no evidence in the literature that chronic hypercapnia is associated with diaphragm fatigue. I'm not talking about exercising the muscles of these patients. All I'm saying is that when you have a hypercapnic, underventilated neuromuscular patient, and you rest his muscles and normalize his arterial gases using inspiratory muscle aids, central nervous system bicarbonate levels normalize, chemotactic centers become more responsive, and the patient can maintain normal blood gases for longer periods of time when breathing on his own. Once again, I use oximetry biofeedback—by keeping my underventilated patients on room air and telling them to maintain normal saturation by breathing on their own (that is, breathing deeper), and when they get tired to use intermittent positive pressure ventilation via a mouthpiece or a nosepiece.

Sortor-Leger: Connected to a ventilator.

Bach: Yes, with a portable ventilator using high inspiratory pressures—high IPAP BiPAP.

Sortor-Leger: OK. I think that's right. There's no question. You ventilate the neuromuscular patient. You normalize their blood gases. By doing that you increase their exercise capacity. You increase their ability to breathe better on their own. There's no question. But this is a discussion about exercise, respiratory muscle work, and training. It's a different discussion and your points about COPD cannot, I don't think, be directly applied to neuromuscular patients.

Celli: I started by saying the literature applies to COPD because there was very little on neuromuscular diseases. Having said that, let me just add that there is enough in-vitro and in-vivo evidence that shows that CO_2 in and of itself may make your respiratory muscles malfunction; so I don't see why the big thing, because if you dropped the CO_2 with the ventilator and brought it back down, your function could improve. And John Bach's point, which I happen to adhere to, is that maybe what we ought to do in the neuromuscular patient is not exercise but just retrain the breathing, which is different from exercise. I wanted that point to come out.

Sorter-Leger: With ventilation.

Celli: To normalize values, maybe you need breathing retraining, together with ventilation, to have the patient learn how to breathe a little better again. If we could achieve slower breathing—you know fatigue is a function of two things: the strength over the reserve, P_I/P_{Imax} tied to the length of contraction, t_I/t_{tot}. If we increase frequency, we may decrease the t_I/t_{tot}, and if we decrease t_I/t_{tot}, we will decrease the fatiguing load. So, using physiologic principles, I think we can answer some of these questions, and I think I'll go to my chair.

Hill: Just an editorial comment on that last discussion. I think it's fair to say that the role of fatigue in the hypercapnic neuromuscular patient is unclear, at best.

Stoller: I would like to represent a minority view to say that it's not abnormal to want to exercise.

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Noninvasive Positive Pressure Ventilation at Home

Patrick Leger MD

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Introduction

Noninvasive intermittent positive pressure ventilation (NIPPV) refers to the technique of augmenting alveolar ventilation without an endotracheal airway. I have observed this technique to be less dangerous, have better acceptance, and usually be more comfortable than the invasive technique, which is positive pressure mechanical ventilation by tracheostomy (TIPPV).

The purpose of this paper is to present the technical aspects of NIPPV and the current indications, highlighted by the results of the literature.

Historical Aspects of NIPPV Development

The use of noninvasive ventilation was first documented in 1969 with a description of mouthpiece IPPV; however, its use had begun over a decade earlier.[1] Nasal IPPV was first described by Rideau[2] and Delaubier,[3] early in the 1980s. They were looking for a method other than tracheostomy by which to ventilate Duchenne muscular dystrophy patients. At about that time, knowledge about breathing disorders during sleep was increasing and nasal continuous positive airway pressure (CPAP) was beginning to be used for treatment of sleep apnea syndrome. With these two events, the ingredients for the extension to NIPPV came together. In 1984, our team became interested in NIPPV because, in spite of the effectiveness of TIPPV, it was difficult to convince patients to pursue this avenue of treatment early, and at that time there were no good alternatives. Mechanical ventilation via negative pressure or positive pressure by tracheostomy had been proven effective for patients with restrictive ventilatory defects but has many drawbacks. Negative pressure ventilation is cumbersome and is associated with upper airway obstruction in some patients. It is often difficult to find the correct tracheostomy tube to allow appropriate ventilation and phonation, and irritation and pain are potential complications. The

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last point to explain the development of NIPPV was represented by the limits of the other therapeutic methods such as long-term oxygen therapy (LTOT) in treating restrictive and COPD patients, some of whom went on to develop severe hypercarbia after several years of treatment. The first international publications on NIPPV date from 1987 when some 6 papers reporting on 32 patients documented successful use of this technique.[4-9] Since 1987 the use of NIPPV has continually grown, and to date there are at least 35 papers published, which include approximately 781 patients—49% presented with neuromuscular disease, 29% with chest-wall disorders, and only 22% with chronic obstructive lung disease (COLD).[4-38] The French multicenter study[38] is the largest study published and includes 276 patients studied over a 3-year period. Because of the large number of patients and the extended period of study, I refer to this paper frequently.

Patient-Ventilator Interfaces for NIPPV

Technical Aspects

Three types of interfaces can be used for NIPPV: the nasal mask (Fig. 1), the mouthpiece (Fig. 2), and the full face mask. In Table 1, the general advantages and disadvantages are listed for each of these groups of devices.[39,40] Nasal masks are the most commonly used interfaces with made-to-measure custom-molded masks being more common in Europe, whereas mass-produced industrial masks are more common in the U.S. Mouthpieces are best indicated for daytime use because they are not obtrusive and allow patients to talk and eat but still

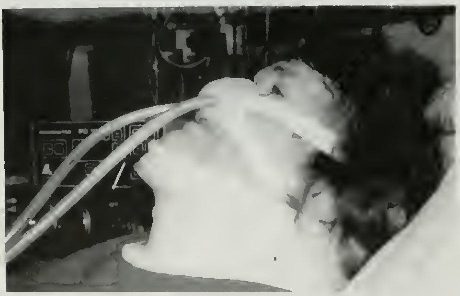

Fig. 1. Nasal mask interface for noninvasive positive pressure ventilation.

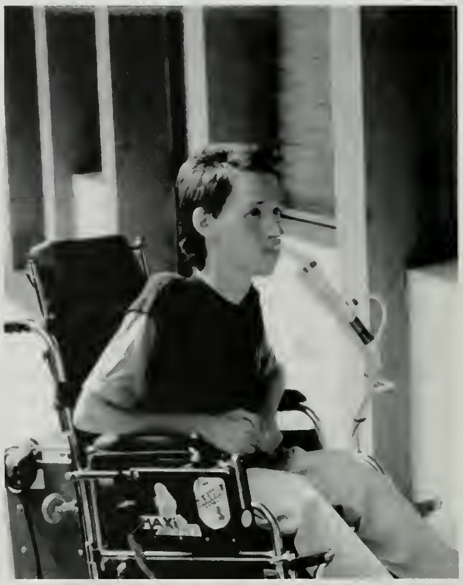

Fig. 2. Mouthpiece interface for noninvasive positive pressure ventilation.

Table 1. Advantages and Disadvantages of Interfaces Used for Noninvasive Positive Pressure Ventilation

Interface	Advantages	Disadvantages
Characteristics common to all interfaces	Physiologic access Industrial or molded	Obtrusive Airway dryness Anchoring problems
Nasal mask	No humidification Stable Mouth free	Irritation to skin, nasal bridge, and mucosa Mouth leaks
Mouthpiece	Better leak control Easy for daytime	Hypersalivation Nasal leaks Orthodontic problems Expectoration impossible
Full face mask	Better leak control	Industrial: poor tolerance
	No anchoring (SONI)	Made to measure: expensive, time-consuming

have the convenience of assisted ventilation when needed.

Criteria for Choosing an Interface

Taking adequate time is essential to choosing the best interface for NIPPV. The main criteria to use are team experience, anatomic considerations, level of ventilator dependence, mode of ventilation to be used, and the efficiency of the first trials. The experience of the medical team with long-term ventilation is important, especially their use of NIPPV. When a team first begins to use NIPPV it may be helpful to designate someone or a group of professionals who can assume the responsibility for choosing, fitting, and making interfaces. Proficiency and efficiency come with experience and practice. This can be achieved quickly if the number of people providing the service is limited. It is our experience that the best results can be achieved by having a variety of interfaces available to offer patients.

The second point is the anatomic considerations of the nose and mouth. To successfully use nasal ventilation a patient must have patent nasal passages. For example, obstruction of the nose because of deviation of the nasal septum is a contraindication for the use of nasal ventilation, and poor dentition can create difficulties for mouth ventilation and/or nasal ventilation.

Another point to discuss is the patient's level of dependence on mechanical ventilation. For nocturnal use, both nasal mask and mouthpiece can be used. Figure 3 shows a patient using a mouthpiece and noseclip for nocturnal ventilation. For longer dependency—more than 12 hours/day—it is necessary to alternate interfaces (for example, nasal mask for stability during the night and mouthpiece for daytime ventilation). The patient's level of physical dependence is also important to consider. Neuro muscular patients with a high degree of paralysis may be unable to don and doff some nasal mask independently. We have found the SONI mask, with its acrylic bite plate and external shell that allow removal by tongue thrust, adaptable to their situation.

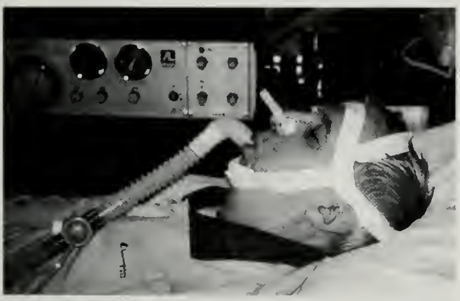

Fig. 3. Patient using mouthpiece and noseclip for nocturnal noninvasive positive pressure ventilation.

The fourth point is the mode of ventilation. For pressure support ventilation with continuous flow, it is frequently possible to use an industrial nasal mask. During volume ventilation, however, the higher peak inspiratory pressure creates more difficulties, and the industrial mask may create skin problems. We therefore typically use the custom-molded mask for volume ventilation.

Effectiveness during the first trials and patient preference are usually the final points to consider in determining which interface will be used at home. Figure 4 shows a patient simultaneously using a nasal mask for ventilation and a mouthstick for communication. In our practice, the first choice is typically a custom-molded nasal mask, the second is the mouthpiece, and if patients need long periods of ventilation during the day both are used and alternated. Full face masks are difficult to use for long-term ventilation at home, and in our opinion only the SONI mask seems suitable for this application.

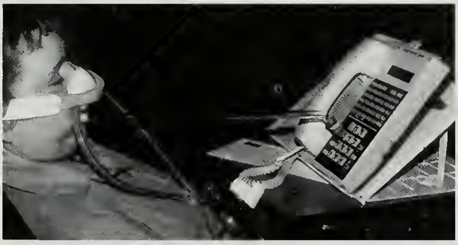

Fig. 4. Tetraplegic patient using nasal mask for noninvasive positive pressure ventilation. Note mouthstick interface with the communications board.

Considerations Related to the Ventilator

Modes of Ventilation

Table 2 compares volume and pressure support ventilators, the two modes of ventilation used for NIPPV. Whereas the volume ventilator delivers a constant predetermined tidal volume, tidal volume delivered by a pressure support ventilator varies according to the preset pressure and the patient's lung and chest-wall compliance. The other main differences that I have observed between the two devices are (1) patient comfort appears greater with pressure support ventilation, primarily related to the sensitive trigger and the absence of an inspiratory peak pressure higher than 22 cm H_2O; and (2) mouth leaks during inspiration are constant during nasal ventilation but their consequences are variable among patients. In theory, pressure support ventilators are able to provide better control of these leaks. Two points could explain this; first is the ability of the device to 'detect' leaks by the variation in the inspiratory flow needed to maintain the preset pressure. The device is able to compensate for the fall in pressure and, in theory, increase patient ventilation even if, in doing so, leaks increase. The second point is the ability of pressure support ventilation to incorporate expiratory positive airway pressure (EPAP). EPAP has not been effective with NIPPV by volume ventilator because of the open system created by the mouth leaks. Pressure support ventilators are able to maintain continuous flow during expiration, creating EPAP. This EPAP may improve the quality of the ventilation and prevent the obstruction of the upper airways, which in turn may explain the oral leaks that occur during inspiration. Because this is still theoretical, it is not possible at this time to make a firm statement that oral leaks can be better prevented or controlled by pressure support ventilation than by volume ventilation.

Table 2. Comparison between Volume and Pressure Support Home Ventilators

Variable	Volume	Pressure Support
Tidal volume	Constant	Variable and limited: maximum IPAP = 22 cm H_2O
Trigger	±	++
Comfort	±	++
Leak control	None	Better ? (↑ flow, EPAP)
Alarm	+	None
Internal battery	+	None
Portability	±	++
Price		
France	++	++ (BiPAP ST™)
USA	+++	++

Criteria for Choosing the Ventilator

In our practice, we can choose between volume and pressure support ventilators and have two criteria for choosing the ventilator with which to begin home mechanical ventilation. The first criterion is the prognosis of the patient and the predicted level of dependency on mechanical ventilation. In many patients who have progressive neuromuscular disease, we can predict that their level of ventilator dependence will increase. They will eventually need to attach the ventilator to their wheelchair and will need an internal battery and alarm. The second criterion is acceptable response to the several days of trials of mechanical ventilation in a monitored setting. During these trials, we monitor the patient's comfort, his synchrony with the ventilator, arterial blood gas values while he is being ventilated, and variables that allow us to determine the quality of nocturnal ventilation. Results allow us to choose the ventilator that is the most suitable for the patient. In our practice, the preference is given to the volume ventilator for (1) progressive neuromuscular patients, (2) patients with low or unstable compliance, (3) patients with hypersecretion, and (4) patients who need to use a mouthpiece. Preference for a pressure support ventilator is given to the nonprogressive neuromuscular patients or other restricted patients with stable compliance and patients who are ambulatory and use only nocturnal ventilation. The difference in price between a volume and pressure support ventilator is not very important in France, but may be important and another deciding factor for choosing a ventilator in other countries.

Ventilator Settings

With NIPPV, conventional ventilator settings are used for volume ventilation, with tidal volume calculated on the basis of 15-20 mL/kg (usually set a little higher than in ventilation without any leaks). The frequency is usually set for 15-20 breaths/min and the I-E ratio is adjusted for 1:1 to 1:3. For pressure support ventilation, we use the BiPAP ST™ system, with a frequency of 15-20 and a minimal expiratory positive airway pressure (EPAP) of 5 cm H_2O as recommended to prevent CO_2 rebreathing. The inspiratory positive airway pressure (IPAP) is adjusted for patient comfort and to correct the CO_2 retention. In our experience, the IPAP level is usually

15-22 cm H_2O. If needed, oxygen supplementation is adjusted for both systems to correct hypoxemia according to the arterial blood gas values and the S_{aO_2} during the nocturnal monitoring, after optimal ventilation has been achieved. In regard to correcting oral leaks, we have had some success by modifying the neck and mandible position to optimize the opening of the upper airways. This is further described later. At discharge, we recommend that the patient use the ventilator at least during all the nocturnal period, and if necessary we increase the duration during the day according to the patient's fatigue level and/or the CO_2 retention. Every attempt is made to achieve the time required for daytime ventilation without disrupting the patient's normal daily activities.

Control of the Effectiveness of NIPPV

We and others[41,42] have found that monitoring during all phases of NIPPV is absolutely necessary to judge the effectiveness of ventilation and to ensure that the patient can return and stay home with security and a well-adapted treatment. The first issue is the comfort of the patient and adaptation to the ventilator during daytime. This is accomplished through multiple awake and nap trials, in the hospital, with adjustments made in ventilator setting and mask fitting. The second point is correction or improvement of arterial blood gas values, which provide the improvement or relief of the symptoms that the patient experienced before the beginning of ventilation. This usually occurs quickly for most patients but, for some, can take time. The third point is the nocturnal controls that are absolutely necessary. For an entire night, we usually record oxygen saturation, ventilator pressure, and/or expired tidal volume. We sometimes measure thoracic and abdominal movement, and, finally, we recommend that arterial blood gas values on ventilation be measured in the early morning (6 AM). Long-term control of effectiveness is essential. Masks need to be adjusted and maintained, especially if the patient has a custom-molded mask. In addition, a patient's status can change over time requiring modifications in management. We monitor stable patients approximately every 6 months after discharge, alternating between an outpatient clinic visit and an inpatient nocturnal recording.

Adverse Effects of NIPPV and Possible Solutions

As can be seen in Table 3, nasal ventilation has some adverse effects.[43] Mask discomfort is a frequent problem and is reported in the literature by as many as 40% of patients. This problem occurs mainly with mass-produced, or industrial, masks. Mask discomfort can be resolved by the use and better fit of different kinds of industrial masks or by using a customized mask. Occasionally, the source of patient discomfort is the ventilator. Resolution should first be attempted by a change in the ventilator settings, but, if discomfort persists, one should not be opposed to trying other machines. Different ventilators have different features that may prove to make the patient more comfortable with NIPPV, such as the control of inspiratory flow or pressure support instead of volume ventilation. Gastrointestinal inflation is another problem and is reported by 50% of patients on volume ventilation. This problem seems to decrease significantly when pressure support ventilators are used. Dryness of the upper airways is reported by about 50% of patients and is usually related to mouth leaks. Controlling

Table 3. Adverse Effects due to Noninvasive Positive Pressure Ventilation and Possible Solutions

Effect	Consequences	Possible Solutions
Traumatic lesions related to interfaces	Skin irritation Nasal or dental pain Dental deformities	Try different interfaces or use molded ones Alternate interfaces during ventilation
Upper airway	Discomfort Decreased secretion clearance	Control mouth leaks Use humidification
Gastrointestinal inflation	Flatulence & pain	Decrease inspiratory peak pressure Try other modes of ventilation
Leakage around the mask or by nose or mouth	Conjunctivitis Decreased efficiency of ventilation Upper airway dryness	Adjust interface Try other interfaces Try chin strap or cervical collar Try other modes of ventilation

the mouth leaks and/or providing humidification usually helps. Humidification is also absolutely necessary in patients with bronchial hypersecretion. Finally, the main complication when using NIPPV is mouth leaks. When patients are nocturnally monitored we find some degree of mouth leaks 100% of the time. The consequences are variable. Sometimes they have no effect on the quality of ventilation, but in other cases they are catastrophic. The adjustment of the ventilator settings with a focus on decreasing inspiratory flowrate, thus decreasing peak inspiratory pressure, often helps. Changing the ventilator could also be tried (such as using a volume ventilator with inspiratory flow control). The oral leaks caused by upper airway obstruction may be resolved by using pressure support ventilation, especially during EPAP. Finally, the use of devices to limit leaks can be very helpful. In our practice, we have had surprising success and patient tolerance with the use of soft cervical collars to limit oral leaks.

Outcomes with NIPPV

The major goals of NIPPV are to control the symptoms of underventilation, to improve the patient's quality of life, and to reduce morbidity and mortality in a cost-effective manner.

Improved ventilation can usually be obtained and was achieved in all the patients we studied who returned home.[38] The results included the elimination of the symptoms of chronic hypoventilation, such as frequent morning headaches that were related to the poor quality of the patients' ventilation at night. The relief of these symptoms was a direct result of the improvement of arterial blood gas values as evidenced by analysis of blood drawn during mechanical ventilation. More impressive was the improvement obtained and maintained during spontaneous breathing on room air. Significant improvement in blood gas values during spontaneous breathing for restrictive patients has been reported in the literature, with an average decrease in P_{aCO_2} of 10 torr and an average improvement in P_{aO_2} of 10 torr. For these patients, the improvement in arterial blood gas values is great enough to allow them to stay on room air during time off of the ventilator. This improvement is maintained over several years, at least 3 years for the majority of the restrictive, scoliosis, and sequelae of tuberculosis patients.[38] The data for COPD patients are more controversial. A randomized crossover study of 19 COPD patients[20] showed no improvement in blood gas values during spontaneous breathing. Uncontrolled studies[23,38] found improvement of P_{aCO_2} and P_{aO_2} after 1 year of NIPPV. In the French retrospective multicenter study,[38] we found a significant improvement in P_{aCO_2}, and this improvement was maintained in Year 1 and Year 2; however, for these patients, the improvement in P_{aO_2} was not great enough to allow them to be free of supplemental oxygen during time off of the ventilator. The exercise capacity is another factor that improved for restrictive patients, but the results are contradictory for COPD patients.[10,19,34] Less impressive were the changes in pulmonary function studies on restrictive and COPD patients, which were nonsignificant .

The quality of sleep is also an important point to review.[5,12,30,38] NIPPV patients, in general, report a subjective improvement in sleep quality despite use of the mask and noise of the ventilator. Patients also report less fatigue during the day. For neuromuscular and restricted patients, this has been confirmed in the literature with objective improvement in sleep quality despite frequent arousals, which were consequences of leaks.

Several studies have assessed patients' perceptions of changes in their symptoms and quality of life following initiation of NIPPV.[5,6,14,17,19,38] In a study of quality of life, which was done by the French Association against Myopathy,[35] 34 boys with Duchenne muscular dystrophy who had used NIPPV for 3 years reported a satisfaction index of 83%. In the French multicenter study,[38] 70% of the patients reported a substantial improvement in their ability to carry out their activities of daily living. This satisfaction index has been reported to be significantly better for restrictive patients than for obstructive patients.

Comparison between hospitalizations before initiation of NIPPV and during the follow-up provides interesting information regarding a patient's medical stability. In the French multicenter study,[38] the changes in the number of days of hospitalization for respiratory illness before and after NIPPV showed a significant reduction for restricted patients (34 days during the year before initiation of ventilation compared to 5 days during Year 1 and Year 2 of mechanical ventilation. The number of hospitalizations for COPD patients decreased significantly only during the first year on NIPPV.

It is possible to evaluate long-term outcome by monitoring the patient's evolution when using NIPPV.[38,44] With time, this evolution proceeds in one of four ways: the patient continues to use NIPPV; the patient stops treatment completely or returns to previous treatment (such as oxygen therapy); the patient stops NIPPV but continues ventilation with a tracheostomy; or the patient dies while using NIPPV. Presented in Figure 5 is the evolution of the 276 patients included in the French multicenter study.[38] Etiologies include 105 scoliosis, 80 sequelae of tuberculosis, 50 COPD, 25 bronchiectasis, and 16 Duchenne muscular dystrophy. This graph shows the percentage (calculated according to the actuarial method) of patients who continue NIPPV, plotted over the years of use. All patients who stopped NIPPV for the reasons mentioned are included in this graph. In summary, scoliosis and patients with sequelae of tuberculosis had the better rate of continuation (after 3 years, 77% of the patients continued NIPPV). These results are similar to those obtained with tracheostomy and mechanical ventilation.[45] The results for COPD are, of course, not as good—with 53% of the patients continuing NIPPV after 3 years; 11 patients dying; and 7 patients needing tracheotomy. It is necessary to understand at this point that the majority of COPD patients included in this study were on LTOT for several years before beginning NIPPV and they presented, despite LTOT, with recurrent acute respiratory failure or poorly tolerated severe hypercarbia. In this situation, NIPPV is an additional technique able to prolong survival of some COPD patients. The two other curves are more surprising. The Duchenne patients had poor results in this group, with about 35% of them continuing NIPPV after 3 years. In fact, 5 patients of the original 16 required tracheotomy and continued mechanical ventilation with this invasive method, and 2 of the original 16 patients died while using NIPPV. The second surprising result is the relatively high percentage of bronchiectasis patients who continued using NIPPV (48% after 3 years). These patients had severe disease when we initiated ventilation. The majority had used LTOT for several years and despite this treatment continued to deteriorate. These results are encouraging for such severe disease.

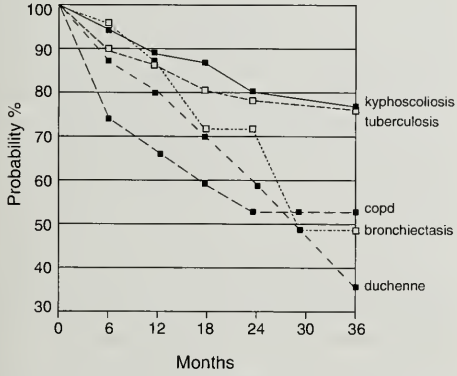

Fig. 5. Probability of 276 patients with specific diseases continuing with noninvasive positive pressure ventilation. (Adapted from Reference 38, with permission.)

Indications for NIPPV

After almost 10 years of work with conditions of various etiologies, we can draw some conclusions regarding the indications for NIPPV.[46] NIPPV is clearly indicated and has been firmly established in patients with chest-wall disorders (such as kyphoscoliosis and deforming sequelae of tuberculosis) and in stable neuromuscular diseases (such as sequelae of poliomyelitis, metabolic myopathy, traumatic tetraplegia, and some congenital myopathies and spinal muscular atrophies). In these patients, NIPPV improves their blood gas values while on ventilation and during time off—in the majority of cases, normalizing P_{aCO_2} and improving P_{aO_2} enough to allow normal daily living without supplemental oxygen. The literature has shown these results to be stable for several years following the initiation of NIPPV. Another argument favoring the use of NIPPV in these patients is the poor results reported when using LTOT alone to treat chronic respiratory insufficiency. Presented in Figure 6 is the study from our group,[47] which shows a comparison of survival rates of scoliosis patients treated by the same team with three different techniques—TIPPV, NIPPV, and LTOT—during the last 10 years (survival curves are calculated by actuarial method). After 3 years, only 60% of kyphoscoliosis patients survived on LTOT, compared to more than 80% on TIPPV and, now, NIPPV. Similar results were obtained for patients with deforming sequelae of tuberculosis. A comparable low survival rate has been reported by Strom et al[48] for scoliosis patients treated on LTOT. These positive results of NIPPV coupled with the poor results with LTOT explain why NIPPV is now the preferred treatment for chronic respiratory insufficiency in this patient population.

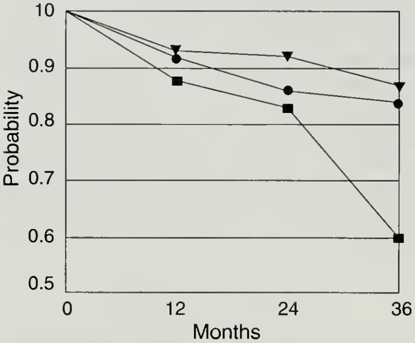

Fig. 6. Comparative survival rates for scoliosis patients receiving mechanical ventilation via tracheostomy (TIPPB, ▼), noninvasive mechanical ventilation (NIPPV, ●), and long-term oxygen therapy alone (LTOT, ■). (Adapted from Reference 47, with permission.)

Not so clearly indicated and less firmly established is the role of NIPPV for patients with progressive neuromuscular disease, such as amyotrophic lateral sclerosis (ALS) and Duchenne muscular dystrophy. Studies have mainly focused on the nocturnal use of NIPPV in these patients; so, it is not surprising that the results are not as good. For these patients, the dependency on mechanical ventilation increases as their disease progresses, resulting in a need to either combine methods of NIPPV (such as mouthpiece during the day and nasal at night) or, if disease progression results in loss of oropharyngeal muscle tone and control of upper airway, tracheostomy ventilation. In addition, these patients need close observation, more attention, and frequent modification of their program of ventilation, especially when NIPPV is used. For these patients NIPPV may be indicated as a step in providing assisted ventilation, but clearly these patients have had and will have less long-term stability.

The group of patients who remain with questionable and controversial indications for NIPPV are the COLD patients. The only currently validated treatment for these patients is LTOT; however, it is difficult to ignore the results of the retrospective study that found in COPD patients a decreased P_{aCO_2}, decreased hospitalizations, and a 53% long-term continuation on NIPPV for 3 years.[38] These data suggest that a subgroup of COPD patients show some improvement when using NIPPV. This subgroup appears to be those patients who (1) are well motivated; (2) were previously well managed by conventional medical treatment and LTOT; (3) have high CO_2 retention; (4) have poor tolerance or insufficient correction of nocturnal S_{aO_2} on oxygen; or (5) are, after acute respiratory failure, unweanable or unable to return to their previous state without the support of NIPPV.

Another group of COLD patients with interesting results are the bronchiectasis and cystic fibrosis patients.[24,32,33] In general, these patients have a poor prognosis, especially when they are inadequately treated with LTOT. Tracheostomy is not usually an acceptable method of delivery for these patients because of their high volume of bronchial secretions. With a tracheostomy, these patients need tracheal suctioning frequently, thus providing them with a poor quality of life and a high risk of systemic infection. NIPPV has been shown to be an interesting treatment alternative—with improved patient comfort and decreased fatigue in general and, especially, during therapy for secretion clearance. The same analysis can be done for patients with cystic fibrosis, especially when these patients are waiting for lung transplantation. It is absolutely clear that NIPPV in the COLD patients remains questionable and controversial. It is also certain that complementary studies are necessary to obtain a precise understanding of the indications for NIPPV in the COLD population.

The final concern is when to initiate NIPPV. There are three choices: (1) during chronic symptomatic hypoventilation, (2) after an episode of acute respiratory failure when the patient cannot be weaned from NIPPV, and (3) prophylactically.

In choosing to initiate NIPPV during chronic symptomatic hypoventilation, the most important point is to be sure that the benefits of treatment, such as the relief of the symptoms, outweigh or support the difficulties of the treatment itself. This is certainly the case in the majority of chest-wall and stable neuromuscular disease patients and is why in our practice they are introduced to NIPPV during chronic symptomatic hypoventilation. For other patients, such as those with COPD, whose benefit of treatment is less certain, NIPPV is more frequently initiated after an episode of acute respiratory failure when the patient is unable to maintain a stable state without continued ventilation.

Despite the initially encouraging results reported from one center,[4] the outcome of a more recent multicenter study on the prophylactic use of nasal ventilation[49] failed to support the contention that the use of nasal ventilation could have any effect on the progression of the restrictive pulmonary syndrome or on the rate of loss of vital capacity for individuals with Duchenne muscular dystrophy.

In Conclusion

Does a role exist for tracheostomy in home mechanical ventilation? The current indications for tracheostomy in home mechanical ventilation include (1) swallowing disorders in neuromuscular patients, (2) ventilator dependence, and (3) ineffective trials of NIPPV due to technical problems in some elective patients. Other than these relatively few indications for tracheostomy, NIPPV, in our practice, is the method of choice for home mechanical ventilation.

REFERENCES

1. Alba A, Solomon M, Trainor FS. Management of respiratory insufficiency in spinal cord lesions. In: Proceedings of the 17th Veteran's Administration Spinal Cord Injury Conference, 1969. Washington DC: U.S. Government Printing Office 0-436-398, 1971:200-213.
2. Rideau Y. Management of the wheel chair muscular dystrophy patient: prevention of death (abstract). In: Proceedings of the 4th International Congress on Neuromuscular Diseases. Los Angeles, 1986.
3. Delaubier A. Traitement de l'insuffisance respiratoire chronique dans les dystrophies musculaires. Memoire pour le Certificat d'études spéciales de rééducation et réadaptation fonctionnelles. Paris: Université René Descartes, 1984:1-124.
4. Delaubier A, Guillou C, Mordelet M, Rideau Y. Assistance ventilatoire précoce par voie nasale dans la dystrophie musculaire de Duchenne. Agressologie 1987;28(7): 737-738.
5. Ellis ER, Bye PT, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular diseases: positive-pressure ventilation through a nose mask. Am Rev Respir Dis 1987;135(1):148-152.
6. Kerby GR, Mayer LS, Pingleton SK. Nocturnal positive pressure ventilation via nasal mask. Am Rev Respir Dis 1987;135(3):738-740.
7. Bach JR, Alba A, Mosher R, Delaubier A. Intermittent positive pressure ventilation via nasal access in the management of respiratory insufficiency. Chest 1987;92(1): 168-170.
8. DiMarco AF, Connors AF, Altose MD. Management of chronic alveolar hypoventilation with nasal positive pressure breathing. Chest 1987;92(5):952-954.
9. Ellis ER, McCauley VB, Mellis C, Sullivan CE. Treatment of alveolar hypoventilation in a six-year old girl with intermittent positive pressure ventilation through a nose mask. Am Rev Respir Dis 1987;136(1):188-191.
10. Carroll N, Branthwaite MA. Control of nocturnal hypoventilation by nasal intermittent positive pressure ventilation. Thorax 1988;43(5):349-353.
11. Segall D. Noninvasive nasal mask-assisted ventilation in respiratory failure of Duchenne muscular dystrophy. Chest 1988;93(6):1298-1300.
12. Ellis ER, Grunstein RR, Chan S, Bye PTB, Sullivan CE. Noninvasive ventilatory support during sleep improves respiratory failure in kyphoscoliosis. Chest 1988;94(4): 811-815.
13. Carroll N, Branthwaite MA. Intermittent positive pressure ventilation by nasal mask: technique and applications. Intensive Care Med 1988;14(2):115-117.
14. Leger P, Jennequin J, Gerard M, Robert D. Home positive pressure ventilation via nasal mask for patients with neuromuscular weakness or restrictive lung or chest-wall disease. Respir Care 1989;34(2):73-79.
15. Bach JR, Alba AS, Shin D. Management alternatives for post-polio respiratory insufficiency: assisted ventilation by nasal or oral-nasal interface. Am J Phys Med Rehabil 1989;68(6):264-271.
16. Rodenstein DO, Stanescu DC, Delguste P, Liistro G, Aubert-Tulkens G. Adaptation to intermittent positive pressure ventilation applied through the nose during day and night. Eur Respir J 1989;2(5):473-478.
17. Heckmatt JZ, Loh L, Dubowitz V. Night-time nasal ventilation in neuromuscular disease. Lancet 1990;335 (8689):579-582.
18. Carrey Z, Gottfried SB, Levy RD. Ventilatory muscle support in respiratory failure with nasal positive pressure ventilation. Chest 1990;97(1):150-158.
19. Goldstein RS, De Rosie JA, Avendano MA, Dolmage TE. Influence of noninvasive positive pressure ventilation on inspiratory muscles. Chest 1991;99(2):408-415.
20. Strumpf DA, Millman RP, Carlisle CC, Grattan LM, Ryan SM, Erickson AD, et al. Nocturnal positive-pressure ventilation via nasal mask in patients with severe chronic obstructive pulmonary disease. Am Rev Respir Dis 1991;144(6):1234-1239.

21. Rigault JY, Leroy F, Poncey C, Brun J, Mallet JF. Ventilation mécanique prolongée par voie nasale: a propos de 27 cas de myopathie. Rev Mal Respir 1991;8(5): 479-485.
22. Elliott MW, Mulvey DA, Moxham J, Green M, Branthwaite MA. Domiciliary nocturnal nasal intermittent positive pressure ventilation in COPD: mechanisms underlying changes in arterial blood gas tensions. Eur Respir J 1991;4(9):1044-1052.
23. Leroyer C, Andre N, Robinet G, Martin F, Dewitte JD, Quiott JJ, et al. Nasal intermittent positive pressure ventilation (NIPPV) in COPD patients: immediate and one year on results (abstract). Am Rev Respir Dis 1991;143 (4, Part 2):A74.
24. Hodson ME, Madden BP, Steven MH, Tsang VT, Yacoub MH. Non-invasive mechanical ventilation for cystic fibrosis patients: a potential bridge to transplantation. Eur Respir J 1991;4(5):524-527.
25. Gay PC, Patel AM, Viggiano RW, Hubmayr RD. Nocturnal nasal ventilation for treatment of patients with hypercapnic respiratory failure. Mayo Clin Proc 1991; 66(7):695-703.
26. Efthimiou J, Butler J, Benson MK, Westaby S. Bilateral diaphragm paralysis after cardiac surgery with topical hypothermia. Thorax 1991;46(5):351-354.
27. Hill NS, Eveloff SE, Carlisle CC, Goff SG. Efficacy of nocturnal nasal ventilation in patients with restrictive thoracic disease. Am Rev Respir Dis 1992;145(2, Part 1):365-371.
28. Goodenberger DM, Couser JI, May JJ. Successful discontinuation of ventilation via tracheostomy by substitution of nasal positive pressure ventilation. Chest 1992; 102(4):1277-1279.
29. Sortor S. Pulmonary issues in quadriplegia. Eur Respir Rev 1992;2(10):330-334.
30. Elliott MW, Simonds AK, Carroll MP, Wedzicha JA, Branthwaite MA. Domiciliary nocturnal nasal intermittent positive pressure ventilation in hypercapnic respiratory failure due to chronic obstructive lung disease: effects on sleep and quality of life. Thorax 1992;47(5): 342-348.
31. Branthwaite MA. Mechanical ventilation for stable COPD: alternative, adjunct or sequel to LTOT. Eur Respir Rev 1992;2(10):346-347.
32. Piper AJ, Parker S, Torzillo PJ, Sullivan CE, Bye PT. Nocturnal nasal IPPV stabilizes patients with cystic fibrosis and hypercapnic respiratory failure. Chest 1992; 102(3):846-850.
33. Bellon G, Mounier M, Guidicelli J, Gerard M, Alkurdi M. Nasal intermittent positive pressure ventilation in cystic fibrosis. Eur Respir Rev 1992;2(10):357-359.
34. Laier-Groeneveld G, Huttemann U, Criée CP. Nasal inspiratory positive pressure ventilation. Eur Respir Rev 1992;2(10):389-397.
35. Paulus J, Willig TN. Nasal ventilation in neuromuscular disorders: respiratory management and patients experience. Eur Respir Rev 1993;3(12):245-250.
36. Bach JR. Management of neuromuscular ventilatory failure by 24 hour non-invasive intermittent positive pressure ventilation. Eur Respir Rev 1993;3(12):284-292.
37. Barois A, Estournet-Mathiaud B. Nasal ventilation in congenital myopathies and spinal muscular atrophies. Eur Respir Rev 1993;3(12):275-279.
38. Leger P, Bedicam JM, Cornette A, Reybet-Degat O, Langevin B, Polu JM, et al. Nasal intermittent positive pressure ventilation: long-term follow-up in patients with severe chronic respiratory insufficiency. Chest 1994;105(1):100-105.
39. Bach JR, Sortor SM, Saporito LR. Interfaces for non-invasive intermittent positive pressure ventilatory support in North America. Eur Respir Rev 1993;3(12):254-259.
40. Cornette A, Mougel D. Ventilatory assistance via the nasal route: masks and fittings. Eur Respir Rev 1993; 3(12):250-254.
41. Langevin B, Leger P, Gerard M, Sukkar F, Guez A, Robert D. Monitoring nasal ventilation. Eur Respir Rev 1993;3(12):260-266.
42. Delguste P, Rodenstein DO. Implementation and monitoring of mechanical ventilation via nasal access. Eur Respir Rev 1993;3(12):266-270.
43. Leger P, Langevin B, Guez A, Sukkar F, Sortor Leger S, Robert D. What to do when nasal ventilation fails for neuromuscular patients? Eur Respir Rev 1993;3(12): 279-284.
44. Simonds AK, Elliott MW, Carroll N, Branthwaite MA. Longterm nasal intermittent positive pressure ventilation: clinical, physiological and economic outcome (abstract). Am Rev Respir Dis 1991;143(4, Part 2):A586.
45. Robert D, Gerard M, Leger P, Buffat J, Jennequin J, Holzapfel L, et al. La ventilation mécanique à domicile définitive par trachéotomie de l'insuffisant respiratoire chronique. Rev Fr Mal Respir 1983;11(6):923-936.
46. Hill NS. Noninvasive ventilation: does it work, for whom, and how? Am Rev Respir Dis 1993;147(4):1050-1055.
47. Robert D, Leger P, Gerard M, Gaussorgues P, Jay S. Utilisation de l'oxygenotherapie de longue duree en dehors des bronchopneumopathies obstructives (BPCO). Agressologie 1988;29(7):525-528.
48. Strom K, Pehrsson K, Boe J, Nachemson A. Survival of patients with severe thoracic spine deformities receiving domiciliary oxygen therapy. Chest 1992;102(1):164-168.
49. Raphaël JC, Chevret S, Chastang C, Bouvet F. Home mechanical ventilation in Duchenne's muscular dystrophy in search of a therapeutic strategy. Eur Respir Rev 1993;3(12):270-274.

Leger Discussion

Bach: I agreed with almost everything you had to say, but I have one or two comments. First of all, the concept of preventive ventilation in this country does not exist. The idea is that if you use nasal ventilation every night, your vital capacity is going to decrease less quickly and it's going to prolong survival. However, patients will continue to weaken. To significantly prolong survival, they're going to need more than just nocturnal nasal ventilation. Yves Rideau insists, however, that, in fact, preventive ventilation does work—and he will soon publish supporting data. Although bias is really against it. The study by the French Muscular Dystrophy Association, which contradicted Rideau, was, in fact, useless. It was a multicenter study with little standardization and no attempt at documenting patient compliance with the protocol. They made no effort to find comfortable nosepieces for the patients. They made no effort to even standardize how they measured vital capacity. So, to use that study to negate preventive ventilation is ridiculous, OK? I just wanted to mention that in passing.

Leger: It's clear that for those patients the compliance was very poor. It is one of the results of the study. These patients had no symptoms, and we proposed a very basic technique that in fact turned out to be difficult and inconvenient to use. The results showed compliance to be very poor because the patients had no symptoms, and they thought it was not necessary to complicate their lives with this kind of device. In my opinion there was less of a need for the treatment.

Bach: But, Patrick, the people who did that study didn't believe in it. Now, Rideau will say that he evaluated patient compliance and he found that his patients who use it 8 hours every night do better. If you don't know how much your patients are actually using the technique, you can't come to a conclusion.

Sortor-Leger: Is that study published?

Bach: Yes, his group's preliminary study was published and another is in preparation.[1]

REFERENCE

1. Delaubier A, Guillou C, Renardel A. Plise en charge de l'insuffisance respiratoire. Cah Kinesither 1988;133:64-72.

Kacmarek: I think all that Patrick is saying is that the data that are available have demonstrated that it may not be useful.

Bach: And I believe that.

Kacmarek: That there needs to be more research in order to clarify the issue.

Hill: First of all, I think you gave an excellent overview of a pretty complicated topic, and I congratulate you, Patrick. Second, I heard Rideau present his data in Boston about 6 years ago, and at that time, he had no controls. Although the data looked promising, without controls it's uninterpretable. I bet he doesn't have controls now.

Bach: No, I think he does now.

Hill: Does he? Well, I'll have to see it. Partly because of his talk, we considered initiating a similar controlled trial in the Boston area, but we got absolutely nowhere because of the same problem Patrick encountered, which was that we couldn't get asymptomatic Duchenne patients to use noninvasive ventilation at all. Because it's so difficult to get patients who have no symptoms to use something that's pretty inconvenient with any regularity. I think it's going to be a very tough study to do. I'd like to make two other comments. First, you mentioned that 50% of your patients have gastric hyperinflation. Our patients using noninvasive ventilation complain of flatulence, but very rarely do they complain about any significant problem with gastric hyperinflation. Second, you mentioned that 100% of your patients have leaks. That's our experience, too, but the significance of these leaks varies a lot from patient to patient. We've been trying to look at this in a systematic way and have found some patients who are not benefitting because they're mouth leaking almost continuously. They go into Stage 1 sleep, their mouths open, and they don't get ventilated. We have other patients who seem to have almost normal sleep, despite having fairly frequent leaks. Then, in the middle, other patients have fairly frequent leaks associated with arousals that may have symptomatic implications. You mentioned that you felt the leaks might be related to upper airway obstruction. Can you explain that?

Leger: When we record the ventilator pressure during sleep, we see an initial high peak pressure, completely abnormal, followed by a drop in pressure with a slight or significant plateau. It is our impression that this represents the inspiratory flow arriving at an obstruction, probably the tongue.

Hill: And where is the leak occurring?

Leger: The path of least resistance is through the mouth. This often requires less pressure than is needed to force the upper airway open. In this scenario, you will usually find continuous and massive leaks during the inspiratory cycle. In another scenario, you will see a high peak pressure followed by a significant pressure plateau, which represents the opening of the upper airway and the delivery of the inspiratory flow. In this second situation the oral leaks are less.

Hill: Most of our patients use the BiPAP device, so we're looking at a pressure-limited system. We don't see any pressure increase before leaking occurs.

Kacmarek: Nick (Hill), you're doing the same thing we are. You're using a fair amount of EPAP in those patients. Correct me if I'm wrong, Patrick, when you see *that* you're primarily using a volume-limited ventilator without any CPAP or PEEP?

Leger: Yes, that is right, we are using volume ventilators. It is not helpful with these patients to use PEEP on a volume ventilator, PEEP is completely inefficient. Interesting would be the possibility of continuous expiratory flow with a portable volume ventilator. The continuous expiratory flow is really the interesting aspect of the BiPAP unit.

Hill: The BiPAP S/T™ delivers time-cycled breaths in addition to spontaneous.

Decker: I wanted to know, for the Duchenne patients, were you using BiPAP or were you using a volume ventilator?

Leger: In the study we used volume ventilators. Our rationale for this decision was that we were mostly working with patients who had progressive disease. I think, however, that you can use what you want, in the beginning depending on your organization and the ability you have to change ventilators when the need presents itself. If you have only one choice for a patient, my advice is to begin immediately with a volume ventilator because with the progression of the disease the patient will need eventually to have a ventilator on the wheelchair, with alarms and an internal battery.

Celli: Well, I have a question and a comment. I think the neuromuscular experience is very good and you have outlined it, and everybody agrees with that. I will turn the issue to COPD. The question I ask has to do with the outcomes. If you take a patient who has COPD who gets intubated—John Heffner just reviewed the data for us and showed that over 90% of those guys will come off the ventilator. The reason why I say that is that it may be in that population that we have to think of aggressive ventilation. In our experience and in Nick Hill's experience in a controlled trial, it doesn't look like noninvasive ventilation is very good. On the other hand, all of us have several (maybe close to 20) patients with COPD with tracheostomy ventilation, and it seems to me that those patients can go home. In your patients, what percentage have COPD, have tracheostomies, and were ventilated positively with tracheostomy? Do you think we should pursue that as an avenue of treatment for people with end-stage COPD who've been tubed and ventilated several times?

Hill: Good question.

Make: Bart (Celli), I think you're comparing different kinds of patients. I think we all have experience with patients who are in the intensive care unit and have a tracheostomy, and then we send them home. But they arrived at home ventilation or long-term ventilation differently than the patient who is ventilated more semi-electively (that is, the patient with a high P_{CO_2}), in whom you initiate nasal ventilation and don't initiate tracheostomy ventilation. Those two types of patients have arrived at home ventilation by two different modes—that is, the emergency need to ventilate and then tracheostomy because they can't wean versus the more semi-elective route.

Kacmarek: Can I make one comment relative to what you said? Many patients that we intubate have COPD and have a difficult time weaning. The reflex is to do a tracheotomy, even though you're over the acute phase of the exacerbation. It seems reasonable to transition these patients to nasal ventilation prior to tracheotomy. This is something we should at least try with every one of those patients. Now, I've not been able to convince people to do it on a regular basis, but we have anecdotally been successful in select patients who we could not wean.

Leger: Bart (Celli), I didn't understand the question you posed.

Celli: My question is—we've been tempted to use noninvasive ventilation believing that with the failing COPDer if we intervene we can prevent intubation. I think home preliminary studies show that it's very good.[1] I know you have some data, Nick (Hill) has some data, and the French group led by Brochard has some data. But if you go into an ICU with a decompensation, you can prevent intubation or perhaps you can wean them to noninvasive ventilation. I wanted you to comment on the approach to take with those COPD patients who've reached that point.

REFERENCE

1. Bott J, Carroll MP, Conway JH, Keilty SE, Ward EM, Brown AM, et al. Randomized controlled trial of nasal ventilation in acute ventilatory failure due

to chronic obstructive airways disease. Lancet 1993;341(8860):1555-1557.

Leger: At this time we systematically try to use noninvasive ventilation in the acute phase for those patients. We provide as much time as possible on ventilation by utilizing and alternating between all the possible interfaces. We provide nasal or mouthpiece ventilation during awake periods and full face mask during sleep. We alternate the interfaces as needed to achieve the best tolerance and comfort for the patient. We have had between 50 and 70% of good results. The good results are the patients who had the initial criteria to be intubated, but who were successfully managed without intubation. Our data are uncontrolled, but the multicenter study by Brochard is a controlled study and he confirms these results. In my opinion it is possible in a large majority of cases to prevent intubation.

Make: Yes, but how many of those patients go home on nasal ventilation when you use the . . .?

Leger: It's not really clear. In our experience it is less than 50%.

Bach: In response to Bart's (Celli) question—I think there are two situations. The most common situation is the COPD patient or intrinsic lung disease patient who has an acute exacerbation. There are now in the literature a number of papers[1,2] that indicate that you can at times use noninvasive ventilation as an alternative to intubation. But these patients as soon as they feel better stop using the ventilator, and they're back 2 months later with another acute exacerbation. That happens often, but we don't know that had they continued ventilator use, the exacerbations might have been avoided. However, there's another situation that is less common, but I've seen it twice now—of a patient who has been intubated 15 to 20 or more times, who is a "do not resuscitate," and who refuses reintubation and becomes increasingly obtunded due to hypercapnia. On two occasions we tried to start patients on nasal ventilation, but they were too obtunded to benefit. They breathed through their mouths, exhaling with pursed lips, and it did them no good. Then my respiratory therapist set them up with a mouthpiece and lipseal for intermittent positive pressure ventilation. The hypercapnia decreased, they woke up a bit, then they fell asleep because they were exhausted, and started using nasal ventilation effectively. They not only survived, but they went home using noninvasive ventilation 24 hours/day. The difference was that they were so advanced that they couldn't walk, so they were in wheelchairs 24 hours/day using ventilation like a neuromuscular patient. And they have done extremely well. So, I think that you can do that for certain patients who have become more compliant for one reason or another. One or two other comments. BiPAP is not the only pressure ventilator. There's the GT and GS Bantams that are available through Lifecare Inc. I've found for several patients that the oxyhemoglobin saturations were better when using the volume ventilators than they were with the BiPAP. I couldn't understand why, but now I think that this may be related to the intermittent obstructions, which you reported in your polysomnographies, Patrick. Another thing you said in presenting the French Muscular Dystrophy Association data,[3] that the nasal ventilation patients had a higher incidence of death than the trached patients. But what Raphaël, the primary investigator, was comparing was nocturnal nasal ventilation to 24 hour/day trach ventilation. The second time I heard him present the same data, he concluded that there was no increase in death on nasal ventilation. So, there's a problem with the data interpretation. Also, probability of death data should be used only if necessary.

REFERENCES

1. Benhamou D, Girault C, Faure C, Portier F, Muir JF. Nasal mask ventilation in acute respiratory failure: experience in elderly patients. Chest 1992;102(3):912-917.
2. Pennock BE, Kaplan PD, Carlin BW, Sabangan JS, Magovern JA. Pressure support ventilation with a simplified ventilatory support system administered with a nasal mask in patients with respiratory failure. Chest 1991; 100(5):1371-1376.
3. Raphaël JC, Chevret S, Chastang C, Bouvet F. Home mechanical ventilation in Duchenne's muscular dystrophy in search of a therapeutic strategy. Eur Respir Rev 1993;3(12):270-274.

Sortor-Leger: Not probability of death, John, probability to continue ventilation.

Bach: Now we know pretty much why neuromuscular patients die. I have yet to see a Duchenne patient with a 30% left ventricular ejection fraction (LVEF) die from cardiac failure. They die from mucus plugging or pneumonia when not treated optimally.[1] They rarely die from other than cardiopulmonary causes. Another thing—I was with Rideau from 1981-1983, and I was there when Rideau had the idea. It's like being there when Galileo got hit on the head with an apple or something.

REFERENCE

1. Bach JR. Pulmonary rehabilitation considerations for Duchenne muscular dystrophy: the prolongation of life by respiratory muscle aids. Crit Rev Phys Rehabil Med 1992;3:239-269.

Pierson: That was Newton. It may have happened to Galileo, too, I suppose.

Bach: I went there to show Rideau how to use mouthpiece ventilation. He said "If you can ventilate through the mouth, why don't you try the nose?" So, Dr Delaubier and I put Foley catheters in my nostrils, inflated the cuffs, and I was attached to the ventilator. Sure enough, it worked. But we didn't think at that time it would be useful for nocturnal ventilation because I thought that the air would leak out of the mouth. We started using it in 1982, but Delaubier's 1984 medical thesis was the first publication on nasal ventilation.[1]

REFERENCE

1. Delaubier A. Traitement de l'insuffisance respiratoire chronique dans les dystrophies musculaires. Memoire pour le Certificat d'études spéciales de rééducation et réadaptation fonctionnelles. Paris: Université René Descartes, 1984:1-124.

Kacmarek: One last comment.

Gilmartin: A practical comment. When you're using nasal ventilation for a patient who is acutely sick or critically ill, it is very time intensive. Many times they may fail because if you don't have a dedicated person with that patient 24 hours a day, they'll fail. I see it again and again in our patient population. Also, Patrick (Leger) you spoke about using end-expiratory pressure on your BiPAP patients to flush out the tubings, and that is a problem. With the Respironics BiPAP™ when we first used it, we found that patients were rebreathing CO_2, unless we used expiratory pressure and higher inspiratory pressures. We measured CO_2 all the way back to the machine. We performed a study using four types of valves: the regular Whisper valve, two modified valves (to produce a larger leak rate), and a nonrebreathing valve. We now use a nonrebreathing valve. I don't think we should use high pressures for a patient just to evacuate CO_2. So, I would rather go for a nonrebreathing valve, which doesn't alter the sensitivity very much. They don't rebreath CO_2, they do better, and then as their disease progresses I have the option to increase the IPAP or EPAP.

Bach: They don't just exhale through the tube, you know. They exhale a great deal through the mouth.

Gilmartin: Oh, yes, they do. The Whisper valve leak rate is not great enough for all patients and their exhaled volume goes down through the tubing. We have measured CO_2 all the way down to the machine.

Hill: They have modified their exhalation valves.

Gilmartin: I use the nonrebreathing valve.

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Indications and Criteria for Decannulation and Transition from Invasive to Noninvasive Long-Term Ventilatory Support

John R Bach MD and Louis R Saporito BA RRT

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Characterization of the Problem

Individuals with global alveolar hypoventilation or total respiratory muscle paralysis have both diminshed vital capacities (VCs) and diminished peak cough expiratory flows (PCEFs). Maintaining both adequate alveolar ventilation and adequate PCEF is necessary for preventing pulmonary complications over the long term for these individuals.[1] Individuals with less than 40% of predicted normal VC often have chronic alveolar hypoventilation (CAH) when supine with, at least, subtle symptoms[2] and nocturnal hypoxemia. With less than 30% of predicted normal VC, they usually require nocturnal noninvasive intermittent positive pressure ventilation (IPPV) to prevent morbidity and mortality, particularly when their primary disease is progressive or if they have experienced recent hospitalizations for respiratory difficulties.[2] The VC and PCEF are

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usually further diminished during otherwise benign respiratory tract infections (RTIs) and following general anesthesia.[3] This can cause acute ventilatory failure and lead to potentially fatal bronchial mucus plugging that, if not treated by the institution of appropriate inspiratory and expiratory muscle aids, may necessitate resorting to intubation and possibly tracheostomy.

Any cooperative, unsedated patient with global alveolar hypoventilation but patency of the upper airway adequate to permit unassisted or assisted PCEF greater than 3 to 4 L/s and bulbar muscle function adequate to make an indwelling gastrostomy unnecessary, can be converted to or managed by noninvasive respiratory muscle aids. Tracheal intubation is unnecessary irrespective of the extent of ventilatory insufficiency or paralysis of respiratory muscle function. Individuals who have sustained spinal cord injury (SCI)[4-7] or poliomyelitis[8,9] or who have other neuromuscular conditions benefit most from these approaches.[10-13] The need for high levels of supplemental oxygen in individuals with intrinsic pulmonary disease can greatly diminish the efficacy of noninvasive respiratory muscle aids during sleep, and obstructive pulmonary conditions characterized by inadequate PCEF often do not permit effective use of these methods.

Inspiratory Assistance Methods: An Historical Perspective

Body Ventilators

Negative pressure body ventilators (including the tank, cuirass, and 'wrap' styles) have been available since the 1830s.[14-16] The Emerson Iron Lungs (JH Emerson Co, Cambridge MA) have been manufactured since 1931. Until 1952, body ventilators were the main devices used to provide up to 24-hour long-term ventilatory support for individuals with respiratory failure.[17] The Rocking Bed (JH Emerson Co, Cambridge MA) is a body ventilator that in rocking the patient acts to move abdominal contents and assist diaphragm excursion and, thereby, effects alveolar ventilation. It has been used predominantly by poliomyelitis and muscular dystrophy patients since 1932.[18] It has long been recognized that these body ventilators, which are only practical for nocturnal use, can be effective for long-term ventilatory support in the institutional or home setting.[8] Although the nocturnal use of body ventilators is associated with obstructive apneas that may cause chronic hypertension,[19] for the majority of long-term users the frequent transient nocturnal oxyhemoglobin desaturations that occur during body ventilator use do not seem to cause clinically significant sequelae.[8]

The intermittent abdominal pressure ventilator (IAPV) is another body ventilator that acts on the abdomen to move the diaphragm and thereby assist alveolar ventilation.[20] Because it is convenient, optimizes appearance, and is effective with the subject erect, it continues to be the method of choice for daytime support for many ventilator-assisted individuals (VAIs) with little or no VC or ventilator-free time.[20]

Tracheostomy

In 1871, Trendelenburg first described the use of a tracheostomy tube and inflated cuff for supporting ventilation during anesthesia.[21] Transoral intubation was described soon afterwards,[22] and tracheostomy use became popular for general anesthesia during World War I.[23] Despite this and the fact that tracheostomy tubes were often placed for airway secretion management in individuals ventilated by body ventilators during the 1940s, tracheostomy tubes were not used for ongoing ventilatory support until the inadequate supply of body ventilators made this necessary during the Danish poliomyelitis epidemic in 1952.[24] Although body ventilators could adequately support alveolar ventilation, VAIs were immobilized by them. Evacuation of respiratory tract secretions was difficult in these patients without a tracheostomy, and the limited access to the body made manually assisted coughing difficult or impossible. Noninvasive IPPV had not yet been described, and mechanical insufflation-exsufflation devices for elimination of airway secretions were not available until after 1953.[25-28] Thus, IPPV via tracheostomy gradually became the standard of care for VAIs after 1952.

Reports subsequently appeared on complications related to tracheostomy and long-term IPPV by tracheostomy.[29,30] Undoubtedly the most important factors that render tracheostomy IPPV users susceptible to potentially fatal pulmonary complications are colonization of the airway by pathogenic bacteria and the destruction of natural-airway mucociliary transport mechanisms—the latter includes the ability to generate thoracoabdominal pressures for expiratory airflows adequate to cough up secretions. Introduction of a suction catheter into the left mainstem bronchus can be difficult or impossible, and suctioning can be inadequate to prevent pulmonary complications in many of these patients.[31] The presence of a tracheostomy tube also stimulates bronchial secretions and necessitates regular bronchial suctioning. Communication and swallowing are impaired and aspiration may occur because of the presence of the indwelling tracheostomy tube.[32] In addition to these difficulties, in many States a tracheostomy is considered an 'open wound.' This may prohibit the VAI from community living because of the need for expensive nursing care for tracheal suctioning. Some schools and places of employment also prohibit individuals with such wounds.

Noninvasive IPPV Methods

IPPV can be delivered noninvasively long term to cooperative patients for as much as 24-hours/day. Although first described in 1969, its use had begun over a decade earlier.[33] Air is delivered to the patient through the mouth,[8,10,11,34,35] nose,[2,36,37] or mouth and nose,[38-40] via appropriate patient-ventilator circuit interfaces.

In the mid-to-late 1950s, many VAIs used mouthpiece IPPV for daytime support to free themselves from confinement in tank ventilators. Alba of our group discovered that as patients napped during the daytime, the mouthpiece stayed in the mouth and alveolar ventilation continued to be adequately assisted.[8] By 1964, a number of individuals had already switched from nocturnal use of body ventilators to nocturnal mouthpiece IPPV.[8,10,35] Mouthpiece IPPV permitted these VAIs to conveniently continue total ventilatory support without being subjected to a tracheostomy. In 1957, small, portable positive pressure ventilators became available when the Bantam (now available from Lifecare, Lafayette CO) was released. This greatly increased the likelihood of patient discharge to the community. With the advent of the Bennett Lipseal (Puritan-Bennett, Boulder CO) in 1969, mouthpiece IPPV could be delivered during sleep with less insufflation leakage and little risk of the mouthpiece falling out of the mouth. Our group has reported on 257 VAIs who have used mouthpiece IPPV for up to 24 hours a day as an alternative to tracheostomy IPPV. Use has been sustained for a mean (SD) of 13.2 (12.2) years, or a total of 2,350 patient-years.[11]

In 1982, as an alternative to mouthpiece IPPV for French muscular dystrophy patients, Delaubier, Rideau, and Bach delivered IPPV via a nasal interface constructed from two Foley catheters.[41] We first described nasal IPPV use during sleep by VAIs with little or no measurable VC or ventilator-free time in 1987.[2,42] Commercially available CPAP masks became available for the treatment of sleep-disordered breathing in 1984 and were soon also used to deliver nasal IPPV. Because these CPAP masks are not always comfortable or sufficiently leak free during nasal IPPV, custom-molded varieties were developed. Three such interfaces are available from Lifecare[37] (SEFAM kit, Lifecare, Lafayette CO) and low-profile, transparent-acrylic, custom interfaces can also be individually constructed from a plaster or silicon moulage.[38] In our experience, only individually prepared, custom nasal interfaces have a patient acceptance rate exceeding 50%.

Strapless oral-nasal interfaces have been used since 1985 but were first described in the medical literature in 1989.[39] These interfaces provide an essentially airtight seal for the delivery of IPPV, with only a simple tongue thrust required to expel them. The bite-plate retention is also important for VAIs living alone who are unable to don straps because of inadequate upper extremity function (Fig. 1).[40] All three noninvasive IPPV methods can be used for daytime or nocturnal ventilatory support.[2,9,11,40] The attributes of noninvasive IPPV interfaces are summarized in the Appendix and the relative effectiveness and desirability of all styles of ventilatory support are in Table 1.

Expiratory Muscle Aids: An Historical Perspective

Adequate expiratory muscle function is critical for clearing airway secretions and bronchial mucus plugs, particularly during respiratory tract infec-

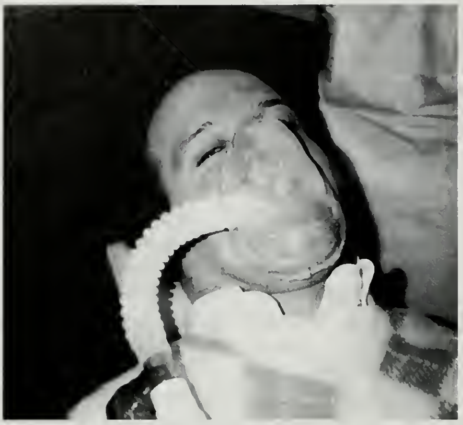

Fig. 1. Subject with spinal cord injury and late-onset chronic ventilatory failure using a strapless oronasal interface for IPPV. (Reproduced with permission of the patient and University of Medicine and Dentistry of New Jersey, Newark NJ.[38])

tions (RTIs), following general anesthesia, and during periods of bronchial hypersecretion for any reason.

A normal cough requires a precough inspiration or insufflation to about 85% to 90% of normal total lung capacity,[43] followed by the development of thoracoabdominal pressures sufficient to generate PCEF exceeding 6 L/s.[44,45] Total expiratory volume, mean (SD), during normal coughing is about 2.3 (0.5) L.[43] Transient glottic narrowing or closure occurs several times before complete expiration.[43] Ventilator users with chronic obstructive pulmonary disease (COPD) have greater degrees of airway narrowing or collapse during coughing and are less likely than ventilator users with neuromuscular disease to be able to generate a PCEF of 5 L/s by unassisted or assisted means.

Manually Assisted Coughing

Techniques of manually assisted coughing involve the use of a manually applied abdominal thrust,

Table I. Effectiveness and Desirability of Methods of Ventilatory Support[a]

	Daytime		Nighttime	
Method	Effectiveness	Desirability	Effectiveness	Desirability
Mouth IPPV	******	******	*	*****
Mouth IPPV[b]	XX	XX	*****	***
Mouth IPPV[c]	XX	XX	******	**
Nasal IPPV	******	**	****	******
ONI IPPV[d]	XX	XX	****	**
SONI IPPV[e]	XX	XX	*****	***
IAPV[f]	***	***	X**	X*
Rocking bed	X*	X****	*	****
Chest shell	X*	X*	**	**
Wrap ventilator	XX	XX	***[g] *[h]	* *
Tank ventilator	XX	XX	****[g] *[h]	* *
GPB[i]	******	******	X	
Trach IPPV[j]	******	*	******	*
Trach IPPV[k]	*****	**	****	**

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[a] Estimates of effectiveness are based on over 500 nocturnal studies of oxyhemoglobin saturation, and desirability is based on a survey of 168 ventilator users who have used both tracheostomy and noninvasive methods. Asterisk is a 1 to 6 scale with 1 low and 6 high; X = occasionally used but not recommended, and XX = should not be used.

[b] Used overnight with a Puritan-Bennett Lipseal.

[c] Used overnight with a Puritan-Bennett Lipseal with cotton pledgets in nostrils covered by tape.

[d] Oronasal interface IPPV with strap retention.

[e] Strapless oronasal interface IPPV.

[f] Intermittent abdominal pressure ventilator, only effective when sitting or semi-recumbent.

[g] For the 40% to 50% of patients without significant sleep obstructive apneas.

[h] For the 50% to 60% of patients with significant to severe sleep obstructive apneas.

[i] Glossopharyngeal breathing (GPB) can be mastered by 50% to 65% of ventilator users with adequate bulbar musculature and used as backup in the event of ventilator failure during sleep.

[j] With an inflated cuff.

[k] With a deflated cuff or cuffless tube.

which may be combined with compression of the anterior chest wall timed to glottic opening.[46] For patients with VCs of less than 1.5 L, the manual assist should be preceded by a deep insufflation. A positive pressure blower (Zephyr, Lifecare, Lafayette CO), intermittent positive pressure breathing (IPPB) machine, or portable ventilator is useful for delivering the deep insufflation. Manually assisted coughing is usually ineffective in the presence of marked scoliosis or obesity and should not be used for 2 hours following meals. Abdominal thrusts should not be used for patients following abdominal trauma or surgery, and anterior chest compression should be avoided in the elderly with osteoporotic ribs. Unfortunately, manually assisted coughing is greatly underutilized.[47] With the prevailing resort to tracheostomy, it is no longer widely taught to healthcare professionals.[44]

Mechanical Insufflation-Exsufflation

When manual assistance is inadequate or contraindicated, the most effective alternative for generating optimal PCEF and clearing airway secretions is the use of mechanical insufflation-exsufflation (MI-E).

In 1951, Barach et al described mechanical exsufflation.[48] They used a vacuum cleaner motor with a 5-inch solenoid valve attached to an iron lung portal. At peak negative pressure in the tank, the valve opened, triggering an exsufflation as intratank pressure became atmospheric in 0.06 seconds.[48] An additional increase in exsufflation flows was obtained by timing an abdominal compression to coincide with valve opening.[49] Investigators[48] reported that the use of these techniques "completely replaced bronchoscopy as a means of keeping the airway clear of thick tenacious secretions.... and another patient would have required bronchoscopy or re-opening of the tracheotomy if the exsufflator had not been successful in clearing the airway."

In 1953, various portable devices, the best known of which was the Cof-flator (OEM Co, Norwalk CT),[25] became available to deliver MI-E directly to the airway via a mouthpiece, mask, or endotracheal tube.[25,50] Insufflation and exsufflation pressures were independently adjusted for comfort and efficacy. The Cof-flator consisted of a 2-stage axial compressor that gradually inflated the lungs over a 2-second period with positive pressure, usually to about 30-40 torr. The pressure in the upper respiratory passageway was then dropped to 30-40 torr below atmospheric pressure in 0.02 seconds by the swift opening of a solenoid valve connected to a negative pressure blower. The negative pressure was usually maintained for 1-3 seconds.[27,51] The Cof-flator went off the market in the mid-1960s when the manufacturer went out of business and tracheal suctioning and bronchoscopy became the standards for airway secretion management.

In February 1993, a new MI-E device (In-Exsufflator, JH Emerson Co, Cambridge MA) became available (Fig. 2). It operates like the Cof-flator except that cycling between positive and negative pressure is done manually. The manual cycling feature facilitates caregiver-patient coordination of inspiration and expiration with insufflation and exsufflation, but it requires a third hand when one wants to deliver a concomitant abdominal thrust or if an additional hand is needed to secure the mask. An abdominal thrust applied during exsufflation increases PCEF.[46] One treatment consists of about 5 cycles of MI-E followed by a period of normal breathing or ventilator use for 20-30 seconds to avoid hyperventilation. Five or more treatments are given in one sitting, and the treatments are repeated until no further secretions are expelled; this regimen can be repeated every 10-15 minutes if necessary.

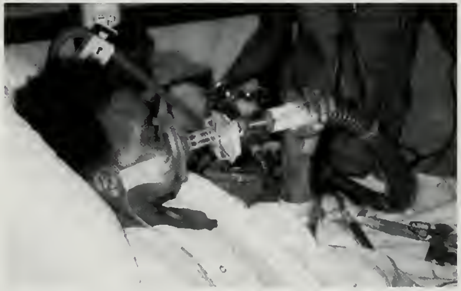

Fig. 2. Myasthenia gravis patient during an acute exacerbation, clearing airway secretions using an In-Exsufflator. (Reproduced with permission of the patient.)

Previously abnormal VC, pulmonary flowrates, and oxyhemoglobin saturation (S_{aO_2}) improve with clearing of mucus plugs by MI-E.[12,44,52] Significant increases in PCEF were demonstrated during MI-E in patients with bronchiectasis, asthma, and pulmonary emphysema.[27] An increase in VC of 15-42% was noted immediately following treatment in 67 patients with "obstructive dyspnea," and a 55% increase in VC was noted following MI-E in congested patients with neuromuscular conditions.[53] We have observed a 15-50% improvement in VC and normalization of S_{aO_2} by MI-E for acutely ill neuromuscular VAIs.[46] The efficacy of MI-E applied by oral-nasal mask has also been demonstrated in animal models.[51] MI-E can be applied via an endotracheal or tracheostomy tube to clear secretions without the airway irritation and discomfort caused by tracheal suctioning, and this was demonstrated to be effective in reversing acute atelectasis in 1954.[54] However, PCEF can be greater when MI-E is applied via a mask in some patients.[55] It is also our clinical impression that transtracheal use of MI-E (rather than suctioning) is associated with a decrease in the production of airway secretions caused by the irritating effects of airway suctioning.

Williams and Holaday[56] reported that MI-E could effectively eliminate airway secretions and ventilate patients in the minutes following general anesthesia. They applied MI-E both to cooperative and to unconscious patients and reported normalization of blood gases in all 7 individuals studied, including 2 with advanced pulmonary emphysema. In addition, improved breath sounds, increased percussion resonance, reduced respiratory rate, clearing of cyanosis, and reversal of right lower-lobe collapse were reported for selected patients as a direct result of MI-E. Patients with wounds of the abdomen or chest have reported less wound pain during MI-E than when coughing spontaneously.[29]

The use of MI-E permits us to extubate neuromuscular patients immediately after general anesthesia for elective surgery despite their having little or no ventilator-free time, and to convert them to the use of noninvasive IPPV. It also permits us to avoid intubation or to quickly extubate neuromuscular patients with profuse airway secretions in acute respiratory failure during RTIs.

No pulmonary complications have been reported with the use of MI-E. Colebatch[57] noted that because the negative pressure applied to the airways is analogous to positive pressure on the surface of the lungs during a normal cough, it is improbable that this negative pressure can be more detrimental to the lungs than the normal cough pressure gradient. Bickerman[51] found no evidence of parenchymal damage, hemorrhage, alveolar tears, or emphysematous blebs in the lungs of animals treated with MI-E. Barach and Beck[53] reported no serious complications in the 103 patients they treated with over 2,000 courses of MI-E, and for no patient did MI-E have to be discontinued. No reports of damaging side effects were disclosed in more than 6,000 MI-E treatments in over 400 patients, most of whom had primarily lung disease.[57,58] Consistent with this is the fact that in over 650 patient-years and hundreds of applications of MI-E by our neuromuscular VAIs, no episodes of pneumothorax, aspiration of gastric contents, or blood-streaking of sputum have been observed.[46] Caution must be observed for bradycardias, and insufflation and exsufflation pressures should be increased gradually when MI-E is used for acute traumatic tetraplegics.[59]

Indications & Criteria for Use of Noninvasive Respiratory Aids

Any individual requiring ventilatory support, either acutely or long term, can be ventilated by noninvasive IPPV as an alternative to endotracheal intubation or tracheostomy, provided these criteria are met: (1) the patient is mentally competent, cooperative, and not using heavy sedation or narcotics; (2) supplemental oxygen therapy is unnecessary or minimal; (3) S_{aO_2} is maintained above 90% by aggressive airway secretion elimination as necessary; (4) bulbar muscle function is adequate for swallowing without potentially dangerous aspiration; (5) no history exists of substance abuse or uncontrollable seizures; (6) unassisted or manually assisted PCEFs exceed at least 3 L/s; and (7) no conditions are present that interfere with the use of IPPV interfaces (eg, facial fractures, inadequate bite for mouthpiece entry, presence of a nasogastric tube or facial hair that might hamper airtight seal with the lipseal or nasal interfaces).

We have observed that users of noninvasive methods of ventilatory support who try both negative pressure body ventilators and noninvasive IPPV methods at the onset of ventilatory failure invariably prefer the latter provided that a comfortable interface can be provided. We, therefore, only resort to the use of negative pressure body ventilators to facilitate tracheostomy site closure following decannulation (for transition from tracheostomy IPPV to noninvasive IPPV); during extubation and transition to noninvasive IPPV for patients with no significant ventilator-free time who are not experienced in using noninvasive IPPV methods; and occasionally during RTIs when nasal congestion interferes with successful use of nasal IPPV and frequent need for suctioning oral secretions makes IPPV by lipseal less practical. Thus, noninvasive IPPV methods are often the only methods of ventilatory support needed to treat ventilatory failure. The patient should be given the opportunity to choose between use of nasal or mouthpiece IPPV, to try a lipseal for nocturnal use, and, possibly, to use an IAPV during daytime hours.

Chronic alveolar hypoventilation develops insidiously. Symptoms are usually subtle and the diagnosis is frequently missed unless specifically pursued.[2] The following indications should be considered when initiating nocturnal noninvasive IPPV in the stable patient: (1) the speed of progression of the illness, (2) the presence of hypercapnia or end-tidal P_{aCO_2} exceeding 50 torr during sleep, (3) nocturnal mean S_{aO_2} less than 95%, and (4) the presence of symptoms of CAH.

We believe that any patient with symptoms suggesting CAH or sleep-disordered breathing should undergo nocturnal S_{aO_2} monitoring and possibly capnography. A VC of less than 40% of predicted normal measured in the supine position is highly indicative of nocturnal hypoventilation from inspiratory muscle weakness, whereas symptomatic patients with greater VCs are more likely to have predominantly central or obstructive sleep apneas with or without severe concomitant inspiratory muscle weakness. We introduce and train these patients to use noninvasive IPPV and MI-E in the clinic and home setting. Subjects with relatively stable neuromuscular conditions such as spinal muscular atrophy or certain patients with congenital myopathies can tolerate mild CAH with mild hypercapnia and mean nocturnal S_{aO_2} of 93-94% for years. They require ventilatory support only during RTIs. Patients with rapidly progressive conditions such as amyotrophic lateral sclerosis (ALS) or frequent hospitalizations for respiratory difficulties should begin nocturnal noninvasive IPPV once nocturnal hypercapnia or prolonged oxyhemoglobin desaturation appears even when symptoms are not obvious. As their neuromuscular conditions progress, the majority of these patients gradually increase their daily regimen of noninvasive IPPV to 24 hours/day without ever being hospitalized. Although most ALS patients will eventually require a tracheostomy tube to prolong survival, the use of noninvasive inspiratory and expiratory muscle aids alone can increase survival and quality of life for many while postponing or eliminating the need to resort to tracheostomy.[13] Tracheostomy should be discouraged for the majority of these patients.

For postpoliomyelitis patients and high-level tetraplegics, and for most advanced neuromuscular patients, initial episodes of acute respiratory failure are usually precipitated by upper respiratory infections. Because these patients may have never used MI-E, noninvasive IPPV, and oximetry biofeedback,[12] we often manage the initial episode by hospitalization for surveillance and patient and caregiver training. Despite dyspnea, hyperpyrexia, leukocytosis, atelectasis as seen by baseline S_{aO_2} in the low 90s during IPPV, and acute hypercapnic respiratory failure, we have consistently managed subsequent episodes without hospitalization by using mouthpiece and/or nasal IPPV, MI-E, and oximetry in the home. Hospitalization is recommended in the presence of clinically important dehydration, severe bulbar muscle dysfunction, radiographic evidence of pulmonary infiltration, progressive disease despite antibiotic therapy, or baseline S_{aO_2} below 90% or P_{aO2} below 50 torr in the presence of eucapnia or hypocapnia. Only in the latter two scenarios should patients receive supplemental oxygen.

Criteria for Tracheostomy Tube Removal

We have managed more than 75 patients with no ventilator-free time by mouthpiece IPPV up to 24 hours/day for a decade or more[11] and have used nocturnal nasal IPPV for others who have little or no measurable VC.[2,42] Nocturnal oximetry studies of the users of mouthpiece IPPV yielded mean S_{aO_2} of 95% or greater in 76% of cases. Therefore, we believe that no level of VC or level of inspiratory muscle dysfunction should be used as an indication for intubation, extubation, tracheostomy, or decannulation.

We are unaware of attempts to establish criteria to permit safe and efficient decannulation or indicate use of noninvasive alternatives to tracheostomy. One of us (JRB) has described the use of the maximum insufflation capacity (MIC), a measure of the maximum volume of air that can be held with a closed glottis before being expelled.[12] MIC is a function of bulbar muscle integrity and is obtained by some combination of air stacking of mechanical insufflations and glossopharyngeal breathing. Weak oral muscles can be splinted during these insufflations by delivering the air via a lipseal held firmly over the mouth. MIC is a function of pulmonary compliance and strength of oropharyngeal and laryngeal musculature. An MIC of at least 1,000 mL is necessary to achieve adequate PCEF during attempts at manually assisted coughing.[12] With an MIC of less than 1,000 mL, regular and effective MI-E may be necessary to avoid hospitalization and intubation during RTIs. With less than 1,000 mL of MIC, however, we do not recommend closure of a tracheostomy for VAIs who have been using tracheostomy IPPV without significant complications. We have suggested that an MIC of less than 500 mL is an indication for tracheostomy.[12]

In our experience, for individuals who are not ventilator assisted and who are not able to engage effectively in glossopharyngeal breathing, the evaluation of MIC is impractical because it necessitates the presence of positive pressure ventilatory equipment. MIC also does not yield sufficient information on the patency of the upper airway. An MIC of 500 mL implies severe ventilatory insufficiency (because the VC can only be less than this amount). However, even individuals with MIC > 1,000 mL who are able to breathe effectively without assistance may not be able to have their tracheostomy tubes safely removed because of difficulty in clearing secretions due to upper airway obstruction.

The ability to effectively clear airway secretions when necessary is dependent on normal function of the glottis and patency of the upper airway. Upper airway obstruction that may not be so severe as to impair autonomous breathing may manifest as diminished PCEF during attempts at assisted and unassisted coughing and may render tracheostomy removal unsafe. Diminished PCEFs are common following intubation in patients who go on to have obstruction due to dysfunctional vocal cords and/or hypopharyngeal collapse. PCEF can be measured using a peak flow meter (eg, Model 710, Health-Scan Products Inc, Cedar Grove NJ) or any digital spirometer that measures expiratory flow. Normal PCEF ranges from 5 to 14 L/s.[43,60] At least 3-4 L/s are necessary for safe tracheostomy tube removal and airway secretion elimination.[44,45] Although few autonomously breathing individuals with SCI and even fewer VAIs can attain 3 L/s without assistance, most can obtain such flows by manually assisted coughing. When manually assisted coughing cannot be used or when it cannot generate optimal PCEFs, MI-E can be used to increase airflows.[44,46] When maximum unassisted or assisted PCEF cannot exceed 3 L/s, the tracheostomy site should not be allowed to close. Other factors that may hamper use of noninvasive aids include the presence of concomitant intrinsic pulmonary disease that necessitates oxygen therapy. Supplemental oxygen can render the use of noninvasive IPPV ineffective by impairing the central nervous system reflexes that are necessary during sleep to prevent excessive insufflation leakage and underventilation.[2]

Ventilator Weaning & How To Convert to Noninvasive Methods

We believe that any VAI who satisfies the criteria noted in the section "Indications and Criteria for Use of Noninvasive Respiratory Aids" is a candidate to have his tracheostomy tube removed and be switched to noninvasive IPPV. Practically speaking, any VAI with a static or slowly progressive condition and sufficiently intact bulbar musculature for potentially effective glossopharyngeal breathing should have his tracheostomy tube removed as part of a routine rehabilitation program. This includes VAIs with SCI, postpoliomyelitis, most non-Duchenne muscular dystrophy myopathies, Type II or III spinal muscular atrophy, and individuals with other relatively stable neuromuscular conditions. Individuals with rapidly progressive conditions such as ALS or typical Duchenne muscular dystrophy may never need to be intubated or undergo tracheostomy and, yet, still benefit from 24-hour ventilatory support,[10,13] but once tracheostomized if they have had no significant difficulties the tracheostomy should be maintained. Such patients are generally poor candidates to achieve ventilator-free time by glossopharyngeal breathing and, therefore, there may be no clear advantage in switching them to noninvasive aids.

At least 40 VAIs with spinal-cord injuries, 50 to 100 postpoliomyelitis VAIs, and many other VAIs with progressive neuromuscular conditions have been converted from tracheostomy IPPV to the 24-hour use of long-term regimens of noninvasive support including IPPV.[4-6,11] The strategy and methods for accomplishing this have been described predominantly for high-level VAIs with spinal-cord injuries.[7] The key steps include (1) placement of a fenestrated cuffed or cuffless tracheostomy tube; (2) removal of any nasogastric tube if present; (3) a brief introduction to mouthpiece and nasal IPPV with the tracheostomy tube capped; (4) tracheostomy tube removal and tracheostomy button placement, usually for 1 or 2 days, with continuous use of noninvasive IPPV; and (5) effective airway secretion clearance by manually or mechanically assisted coughing via a mouthpiece or oronasal mask.

Once adequate nocturnal S_{aO_2} using noninvasive IPPV has been documented,[7,11] the next key step is to remove the button and allow the site to close. If the VAI has more than 2 minutes of ventilator-free time, this can usually be conveniently accomplished using strictly noninvasive IPPV. However, a pressure dressing needs to be applied over the tracheostomy site (Fig. 3). We try to avoid using any dressing taped to the skin because adequate counter pressure is difficult to obtain and the tape can be irritating. Petrolatum gauze is used to plug and cover the stoma. A transparent bio-occlusive dressing secured by tincture of benzoin covers the surrounding skin and makes leakage apparent. Padded dressings are then taped together to form a collar to protect the skin from an outer collar made from 2-in bands of elastic and fitted tightly with a safety pin or Velcro®. Bands of 1-in wide elastic are attached to the elastic collar with safety pins or Velcro®. These bands descend, cross anteriorly under the neck, go under the axillae, ascend to cross over the back, run over the shoulders, and reinsert into the elastic band in front. Padded dressings are folded over the elastic bands to protect the axillae. A roll of tape, a cleaved ping pong ball, or other material can be inserted under the elastic at the stoma to exert additional pressure. This is particularly useful for deeply inset tracheostomy sites and for patients using noninvasive IPPV during stomal closure. Temporary use of a negative pressure body ventilator provides adequate alveolar ventilation while facilitating tracheostomy site closure in the patient with less ventilator-free time.

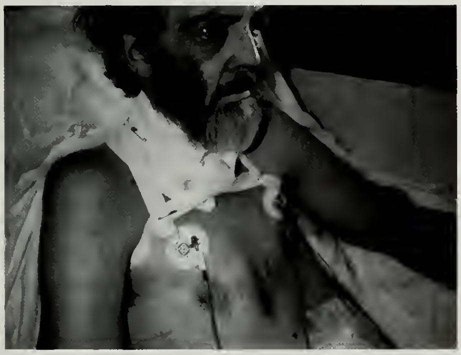

Fig. 3. Occlusive tracheostomy site pressure dressing for VAIs using noninvasive IPPV during closure. (Reproduced with permission of the patient.)

If nasal IPPV is used, the final key step involves trials of various nasal interfaces to optimize seal and comfort. If mass-produced CPAP masks are not adequate, custom-molded varieties should be tried.[38,61] With nocturnal IPPV by mouthpiece, lipseal retention should be used.

An occasional patient who uses the mouthpiece for ventilatory support during daytime hours will want to continue mouthpiece IPPV without a lipseal during sleep. Although we do not recommend this, it may be permitted provided that surveillance is adequate, oximetry alarms are functioning, and the patient has little tendency to lose his mouthpiece.

During the 24-hour use of noninvasive IPPV, the patient is taught to observe and understand the importance of his S_{aO_2}. If weaning is feasible, he weans himself by taking fewer assisted breaths according to how he feels. With this method, no weaning schedule is imposed on the patient. The anxiety inherent in standard weaning approaches that use synchronized intermittent mandatory ventilation, pressure support ventilation, positive end-expiratory pressure, an increase in ventilator-free periods/CPAP, or some combination of these (most often with supplemental oxygen), is avoided because the patient knows that he can take a breath anytime he feels the need.[62]

Occasionally, particularly when negative pressure body ventilators are not used during tracheostomy site closure, the stoma requires several sutures for complete occlusion. Once the stoma is closed, the patient should be taught glossopharyngeal breathing. With the proper teaching technique and spirometric biofeedback,[35,63,64] about 60% of patients learn the technique adequately to free themselves from ventilatory support for useful periods of time (Table 2). Oximetry and, possibly, capnography biofeedback should also be used to adjust ventilator settings, assist the patient who has some ventilator-free time in determining an appropriate daytime schedule of ventilatory assistance, and indicate to caregivers when the patient needs to have airway secretions removed or the ventilation interface adjusted to be more airtight.[7,12]

Table 2. Uses of Glossopharyngeal Breathing

For backup in the event of ventilator failure
For increased ventilator-free breathing time
For ventilatory support during transfer between different methods of noninvasive ventilatory support
For a deeper breath to improve cough effectiveness
To increase speech volume or to shout
To normalize the volume and rhythm of speech
To improve or maintain pulmonary compliance to prevent microatelectasis

Cost and Quality of Life Issues

Total ventilatory support by noninvasive IPPV methods facilitates home management by simplifying and greatly reducing cost. For tracheostomized patients, disposable suction catheters alone can cost hundreds of dollars per month. Tracheostomy tube replacements or sterilization, dressing changes, and often home nursing care, itself, can also be eliminated. A realistic comparison can also be made between the costs of electrophrenic respiration, and the use of noninvasive aids. The initial cost of pacemaker placement and training commonly exceeds $300,000 in the United States especially when placement and training cannot be accomplished in 1 month or less.[7] Longer periods are usually required. Noninvasive aids provide more uniform and often better alveolar ventilation than electrophrenic respiration, without the presence of unphysiologic indwelling foreign bodies.[30] This is because with electrophrenic respiration often one phrenic nerve is stimulated at a time. This causes paradoxic contralateral diaphragm movement (Pendelluft breathing) and often suboptimal alveolar ventilation. Noninvasive aids also eliminate the difficulties associated with tracheostomy and permit ventilator-free breathing time by use of glossopharyngeal breathing.[5] To provide noninvasive IPPV one must obtain a mouthpiece for $18 (Puritan-Bennett Co, Boulder CO). Lipseals and generic nasal interfaces generally cost under $100. Individually molded nasal interfaces cost from $500 to $700[38] and IAPV belts about $350 (Lifecare International Inc, Lafayette CO). Transition from tracheostomy IPPV to noninvasive IPPV generally takes 5 to 10 days in a rehabilitation facility. The ventilator rental and purchase costs are the same irrespective of method used. Further, despite recent improvements in the technology and the enormous cost, electrophrenic respiration is still most often suboptimally effective or of only short-term utility.[30] Because severe obstructive apneas can occur when phrenic nerve stimulation is used during sleep, a cannulated tracheal stoma must be maintained. If the stoma is closed and noninvasive IPPV methods of ventilatory support are used during sleep in these patients, the patients often continue to use noninvasive methods for daytime support and abandon any use of the pacemaker.[30] In our experience, the only indications for electrophrenic pacing are for the ventilatory support of pediatric high-level SCI patients, severe pediatric central alveolar hypoventilation patients, and adult high-level SCI patients with no measurable vital capacity and no neck control—ie, patients who cannot optimally use a mouthpiece.

Quality of life is greatly enhanced by transition to the use of noninvasive ventilatory support methods. In a recent study from our facility,[65] 168 VAIs with long-term experience in using both tracheostomy and noninvasive ventilatory aids overwhelmingly preferred the latter overall and for convenience, safety, speech, appearance, and comfort. The presence of patient-managed attendant care services depends on the absence of so-called open wounds to eliminate the need for expensive home nursing care.[66] We have managed VAIs who could not be returned to the community until they were converted to noninvasive ventilatory support methods.

Difficulties in Initiating the Use of Noninvasive Aids

Despite patient and caregiver preferences for noninvasive approaches,[65] their efficacy for ventilatory support and secretion management, their safety, cost advantages, and the possibility of eliminating hospitalizations, intubations, and bronchoscopies, it has been difficult for physicians and medical centers in the United States to initiate their use. We have observed that noninvasive approaches are not currently part of most health professions school curricula and believe that current invasive approaches are consistent with the general tendency to resort to the highest-level and most expensive technology. Physician and hospital reimbursement is procedure oriented and directed towards inpatient management rather than preventive care. One can be remunerated for hospitalizing a patient, for endotracheal intubation, for tracheostomy, and for bronchoscopy, but outpatient management that avoids these is not understood and often not recognized by third-party payers. Likewise, there is little or no financial incentive for physicians to remove a tracheostomy. We have found that few if any hospital personnel are knowledgeable about mechanical insufflation-exsufflation or familiar with the variety of custom interfaces that can be crucial for optimizing effectiveness and ventilator-user comfort long term. The inevitable sounding of the expiratory volume alarm of the typical hospital ventilator makes it difficult (if not impossible) to use the open systems of noninvasive IPPV in this setting, and many hospitals still do not permit the use of portable ventilators that do not have this alarm. Further, the initial use of noninvasive inspiratory aids requires patience on the part of the caregiver and time for evaluating various masks and other interfaces to optimize comfort and effectiveness. Care is also required to securely maintain the retention straps and to monitor oximetry, particularly during sleep.

There is also a tendency to resort to noninvasive methods too late or only for patients with severe pulmonary pathology. These techniques usually fail if high F_{IO_2}s are required or if the patient is not cooperative.[67] Hence, many clinicians who initially try noninvasive aids in the intensive care setting with patients receiving oxygen are frustrated. We have further observed that nocturnal noninvasive ventilatory support is more successful when the patient's alveolar ventilation is maintained at normal levels with or without ventilatory assistance during daytime hours.[2] Thus, instead of the common practice of providing supplemental oxygen for the underventilated or airway secretion-encumbered patient, noninvasive IPPV methods should be used to normalize blood gases. MI-E should be used to increase pulmonary volumes and reverse oxyhemoglobin desaturations due to mucus plugging, before pneumonia or severe oxygenation impairment necessitates administration of supplemental oxygen.

Yet another difficulty is the small number of global alveolar hypoventilation patients seen by pulmonologists, physiatrists, and respiratory therapists. Neuromuscular patients are rarely referred before an episode of acute respiratory failure has led to their being intubated. This is in part because less than 1% of the more than 220 Jerry Lewis Muscular Dystrophy Association clinics in the United States have pulmonologists as co-directors, and only 18% have physiatrists in this role. The neurologists and other physicians who usually direct these clinics have little if any knowledge of or appreciation of the potential of noninvasive respiratory muscle aids.[68]

In Conclusion

Certainly, our experience with the neuromuscular patient has been consistent with Colebatch's early statement:[57] "As experience with exsufflation with negative pressure increased, bronchoscopy was performed less frequently for the removal of bronchial secretions. At times, bronchoscopic aspiration did relieve acute obstructive anoxia; but it is clear from the case reports that bronchoscopy contributed little to the overall control of bronchial secretions. . . . Had not artificial coughing been available, death might well have followed bronchoscopy in [several cases]. The only possible value of bronchoscopy is to relieve obstruction due to secretions in the trachea and main bronchi. This is usually only of transient benefit, and as the relief can be more easily achieved with exsufflation with negative pressure, bronchoscopic aspiration should rarely, if ever, be performed in patients undergoing artificial respiration." For some VAIs, use of MI-E is lifesaving and pivotal in permitting the long-term use of 24-hour noninvasive ventilatory support. The recent literature has demonstrated the effectiveness of noninvasive approaches primarily for the alveolar hypoventilation patient. Further exploration of their use by VAIs with obstructive or intrinsic pulmonary disease is warranted.

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REFERENCES

1. King M, Brock G, Lundell C. Clearance of mucus by simulated cough. J Appl Physiol 1985;58(6):1776-1782.
2. Bach JR, Alba AS. Management of chronic alveolar hypoventilation by nasal ventilation. Chest 1990;97(1):52-57.
3. Mier-Jedrzejowicz A, Brophy C, Green M. Respiratory muscle weakness during upper respiratory tract infections. Am Rev Respir Dis 1988;138(1):5-7.
4. Bach JR, Alba AS. Noninvasive options for ventilatory support of the traumatic high level quadriplegic patient. Chest 1990;98(3):613-619.
5. Bach JR. New approaches in the rehabilitation of the traumatic high level quadriplegic. Am J Phys Med Rehabil 1991;70(1):13-19.
6. Viroslav J, Sortor S, Rosenblatt R. Alternatives to tracheostomy ventilation in high level SCI (abstract). J Am Paraplegia Soc 1991;14:87.
7. Bach JR. Alternative methods of ventilatory support for the patient with ventilatory failure due to spinal cord injury. J Am Paraplegia Soc 1991;14(4):158-174.
8. Bach JR, Alba AS, Bohatiuk G, Saporito L, Lee M. Mouth intermittent positive pressure ventilation in the management of postpolio respiratory insufficiency. Chest 1987;91(6):859-864.
9. Bach JR, Alba AS, Shin D. Management alternatives for post-polio respiratory insufficiency: assisted ventilation by nasal or oral-nasal interface. Am J Phys Med Rehabil 1989;68(6):264-271.
10. Bach JR, O'Brien J, Krotenberg R, Alba AS. Management of end stage respiratory failure in Duchenne muscular dystrophy. Muscle & Nerve 1987;10(2):177-182.
11. Bach JR, Alba AS, Saporito LR. Intermittent positive pressure ventilation via the mouth as an alternative to tracheostomy for 257 ventilator users. Chest 1993;103 (1):174-182.
12. Bach JR. Pulmonary rehabilitation considerations for Duchenne muscular dystrophy: the prolongation of life by respiratory muscle aids. Crit Rev Phys Rehabil Med 1992;3:239-269.
13. Bach JR. Amyotrophic lateral sclerosis: communication status and survival with ventilatory support. Am J Phys Med Rehabil 1993;72(6):343-349.
14. Dalziel J. On sleep and an apparatus for promoting artificial respiration. Br Assoc Advancement Sci 1838;2:127.
15. Woollam CHM. The development of apparatus for intermittent negative pressure respiration (1) 1832-1918. Anaesthesia 1976;31(4):537-547.
16. Bach JR, Beltrame F. Alternative methods of ventilatory support. In: Rothkopf MM, Askanazi J, eds. Home intensive care. Baltimore: Williams & Wilkins, 1992:173-197.
17. Emerson JH, Loynes JA. The evolution of iron lungs: respirators of the body-encasing type. Cambridge MA: JH Emerson Co, 1978.
18. Eve FG. Actuation of inert diaphragm by gravity method. Lancet 1932;2:995-997.
19. Bach JR, Penek J. Obstructive sleep apnea complicating negative-pressure ventilatory support in patients with chronic paralytic/restrictive ventilatory dysfunction. Chest 1991;99(6):1386-1393.
20. Bach JR, Alba AS. Intermittent abdominal pressure ventilator in a regimen of noninvasive ventilatory support. Chest 1991;99:630-636.
21. Trendelenburg F. Beitrage zur den Operationen an den Luftwagen 2. Tamponnade der Trachea. Arch Klin Chir 1871;12:121-233.
22. MacEwen W. Clinical observations on the introduction of tracheal tubes by the mouth instead of performing tracheotomy or laryngotomy. Br Med J 1880;2:122-124.
23. Magill IW. Development of endotracheal anesthesia. Proc R Soc Med 1928;22:83-88.
24. Lassen HCA. The epidemic of poliomyelitis in Copenhagen, 1952. Proc R Soc Med 1954;47:67-71.
25. The OEM Cof-flator Portable Cough Machine. St Louis MO: Shampaine Industries Inc.
26. Barach AL, Beck GJ, Bickerman HA, Seanor HE. Physical methods simulating cough mechanisms. JAMA 1952;150:1380-1385.
27. Barach AL, Beck GJ, Smith RH. Mechanical production of expiratory flow rates surpassing the capacity of human coughing. Am J Med Sci 1953;226:241-248.
28. Bickerman HA, Itkin S. Exsufflation with negative pressure. Arch Intern Med 1954;93:698-704.
29. Bellamy R, Pitts FW, Stauffer ES. Respiratory complications in traumatic quadriplegia: analysis of 20 years' experience. J Neurosurg 1973;39(5):596-600.
30. Bach JR, O'Connor K. Electrophrenic ventilation: a different perspective. J Am Paraplegia Soc 1991;14(1):9-17.
31. Fishburn MJ, Marino RJ, Ditunno JF Jr. Atelectasis and pneumonia in acute spinal cord injury. Arch Phys Med Rehabil 1990;71(3):197-200.
32. Bonanno PC. Swallowing dysfunction after tracheostomy. Ann Surg 1971;174(1):29-33.
33. Alba A, Solomon M, Trainor FS. Management of respiratory insufficiency in spinal cord lesions. In: Proceedings of the 17th Veteran's Administration Spinal Cord Injury Conference, 1969. Washington DC: U.S. Government Printing Office 0-436-398, 1971:200-213.
34. Curran FJ, Colbert AP. Ventilator management in Duchenne muscular dystrophy and postpoliomyelitis syndrome: twelve years' experience. Arch Phys Med Rehabil 1989;70(3):180-185.
35. Bach JR, Alba AS, Bodofsky E, Curran FJ, Schultheiss M. Glossopharyngeal breathing and noninvasive aids in the management of post-polio respiratory insufficiency. Birth Defects 1987;23(4):99-113.
36. Ellis ER, Bye PTP, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with

neuromuscular disease: positive-pressure ventilation through a nose mask. Am Rev Respir Dis 1987;135(1): 148-152.

37. Leger P, Jennequin J, Gerard M, Robert D. Home positive pressure ventilation via nasal mask for patients with neuromuscular weakness or restrictive lung or chest-wall disease. Respir Care 1989;34(2):73-79.
38. McDermott I, Bach JR, Parker C, Sortor S. Custom-fabricated interfaces for intermittent positive pressure ventilation. Int J Prosthodont 1989;2(3):224-233.
39. Ratzka A. Uberdruckbeatmung durch Mundstuck. In: Frehse U, ed. Spatfolgen nach Poliomyelitis: Chronische Unterbeatmung und Moglichkeiten selbstbestimmter Lebensfuhrung Schwerbehinderter. Munchen, West Germany: Pfennigparade eV, 1989;5:149.
40. Bach JR, McDermott IG. Strapless oral-nasal interface for positive-pressure ventilation. Arch Phys Med Rehabil 1990;71(11):910-913.
41. Delaubier A. Traitement de l'insuffisance respiratoire chronique dans les dystrophies musculaires. In: Memoires de certificat d'etudes superieures de reeducation et readaptation fonctionnelles. Paris: Universite R Descarte, 1984:1-124.
42. Bach JR, Alba A, Mosher R, Delaubier A. Intermittent positive pressure ventilation via nasal access in the management of respiratory insufficiency. Chest 1987;92(1): 168-170.
43. Leith DE. Cough. In: Brain JD, Proctor D, Reid L, eds. Lung biology in health and disease: respiratory defense mechanisms, Part 2. New York: Marcel Dekker, 1977: 545-592.
44. Bach JR, Smith WH, Michaels J, Saporito L, Alba AS, Dayal R, Pan J. Airway secretion clearance by mechanical exsufflation for post-poliomyelitis ventilator-assisted individuals. Arch Phys Med Rehabil 1993;74(2):170-177.
45. Fugl-Meyer AR, Grimby G. Ventilatory function in tetraplegic patients. Scand J Rehabil Med 1971;3(4): 151-160.
46. Bach JR. Mechanical insufflation-exsufflation: comparison of peak expiratory flows with manually assisted and unassisted coughing techniques. Chest 1993;104(5): 1553-1562.
47. Sortor S, McKenzie M. Toward independence: assisted cough (video). Dallas: BioScience Communications of Dallas Inc, 1986.
48. Barach AL, Beck GJ, Bickerman HA, Seanor HE. Mechanical coughing: studies on physical methods of producing high velocity flow rates during the expiratory cycle. Trans Assoc Am Physicians 1951;64:360-363.
49. Barach AL, Beck GJ, Bickerman HA, Seanor HE, Smith W. Physical methods simulating mechanisms of the human cough. J Appl Physiol 1952;5:85-91.
50. Segal MS, Salomon A, Herschfus JA. Alternating positive-negative pressures in mechanical respiration (the cycling valve device employing air pressures). Dis Chest 1954;25:640-648.
51. Bickerman HA. Exsufflation with negative pressure: elimination of radiopaque material and foreign bodies from bronchi of anesthetized dogs. Arch Intern Med 1954;93:698-704.
52. Bach JR. Mechanical exsufflation, noninvasive ventilation and new strategies for pulmonary rehabilitation and sleep disordered breathing. Bull NY Acad Med 1992; 68(2):321-340.
53. Barach AL, Beck GJ. Exsufflation with negative pressure: physiologic and clinical studies in poliomyelitis, bronchial asthma, pulmonary emphysema and bronchiectasis. Arch Intern Med 1954;93:825-841.
54. Beck GJ, Barach AL. Value of mechanical aids in the management of a patient with poliomyelitis. Ann Intern Med 1954;40:1081-1094.
55. Beck GJ, Graham GC, Barach AL. Effect of physical methods on the mechanics of breathing in poliomyelitis. Ann Intern Med 1955;43:549-566.
56. Williams EK, Holaday DA. The use of exsufflation with negative pressure in postoperative patients. Am J Surg 1955;90:637-640.
57. Colebatch HJH. Artificial coughing for patients with respiratory paralysis. Australasian J Med 1961;10:201-212.
58. Barach AL. The application of pressure, including exsufflation, in pulmonary emphysema. Am J Surg 1955;89: 372-382.
59. Bach JR. Update and perspectives on noninvasive respiratory muscle aids: part 2—the expiratory muscle aids. Chest (in press).
60. Leiner GC, Abramowitz S, Small MJ, Stenby VB, Lewis WA. Expiratory peak flow rate: standard values for normal subjects. Am Rev Respir Dis 1963;88:644.
61. Bach JR, Sortor S, Saporito LR. Interfaces for non-invasive intermittent positive pressure ventilatory support in North America. Eur Respir Rev 1993;3:254-259.
62. Bach JR, Alba AS. Tracheostomy ventilation: a study of efficacy with deflated cuffs and cuffless tubes. Chest 1990;97(2):679-683.
63. Dail C, Rodgers M, Guess V, Adkins HV. Glossopharyngeal breathing manual. Downey CA: Professional Staff Assoc of Rancho Los Amigos Hospital Inc, 1979.
64. Dail CW, Affeldt JE. Glossopharyngeal breathing (video). Los Angeles: Department of Visual Education, College of Medical Evangelists, 1954.
65. Bach JR. A comparison of long-term ventilatory support alternatives from the perspective of the patient and care giver. Chest 1993;104(4):1702-1706.
66. Bach JR, Intintola P, Alba AS, Holland IE. The ventilator-assisted individual: cost analysis of institutionalization vs rehabilitation and in-home management. Chest 1992;101(1):26-30.
67. Wysocki M, Tric L, Wolff MA, Gertner J, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure. Chest 1993; 103(3):907-913.
68. Bach JR. Ventilator use by muscular dystrophy association patients: an update. Arch Phys Med Rehabil 1992; 73(2):179-183.

APPENDIX

Description, Advantages, and Disadvantages of Interfaces for Noninvasive IPPV

Mouthpiece IPPV

Interface: Mouthpiece
Description: Simple mouthpiece attached to ventilator tube, straight or flexed varieties. For daytime use, it is either held or for individuals without upper extremity function kept in the mouth or fixed onto motorized wheelchair controls adjacent to the mouth for insufflation as needed.
Advantages: Simple, inexpensive, comfortable, commercially available, used predominantly for daytime IPPV but has been used by some during sleep.
Disadvantages: Long-term use causes orthodontic deformity in some users; occasional allergy to the plastic; may render mouthstick activities less efficient for users with no vital capacity.

Interface: Bennett Lipseal with mouthpiece (available from Puritan-Bennett Inc, Boulder CO)
Description: Plastic flange over the mouth retained by two straps or head-gear. The Lipseal sets the mouthpiece in the mouth and seals the lips for IPPV during sleep.
Advantages: The Lipseal assures firm mouthpiece retention and decreases or eliminates insufflation leak out of the mouth during sleep. A flexible scuba or other soft mouthpiece that accommodates bite may be substituted for the mouthpiece in the Lipseal for additional comfort. System pressures of 30 to 40 cm H_2O can be maintained with this system. In the occasional patient for whom excessive nocturnal nasal insufflation leak cannot be compensated by increasing the delivered ventilator volumes or using pressure-triggered ventilators, cotton pledgets may be placed into the nostrils and sealed in place by tape.
Disadvantages: Difficulty speaking with the Lipseal in place; may increase oral secretions and must be removed to clear oral secretions during respiratory tract infections; adequate seal may be difficult in patients with facial hair; allergy to the plastic; aspiration possible in the event of vomiting.

Interface: Custom-Molded Mouthpiece (available through the Department of Physical Medicine and Rehabilitation at the New Jersey Medical School, Newark NJ)
Description: A firm custom-molded plastic lipseal retained by a bite plate, with or without metal clasps.
Advantages: The interface is removed by tongue thrust without use of the hands; insufflation pressures exceeding 60 cm H_2O can be maintained when necessary.
Disadvantages: The bite plate may stimulate excessive oral secretions or gag reflex in some; its use is dependent on adequate and stable dentition; expensive to fabricate.

Nasal IPPV

Interface: Commercially Available CPAP Masks (Continuous Positive Airway Pressure masks available from Respironics Co Inc, Murrysville PA; Puritan-Bennett; Lifecare International Inc; and others)
Description: Plastic nasal seals or nasal pillows to deliver IPPV, retained by headgear or strap assembly.
Advantages: Simple, inexpensive, durable, easily acquired.
Disadvantages: Often uncomfortable and inadequate nasal seal at the higher pressures required for IPPV.

Interface: SEFAM Mask (available through Lifecare Inc, Lafayette CO)
Description: An assembly kit contains elastic straps, small plastic nasal housing, and silicone putty that can be molded to create 3 to 6 interfaces. The three styles available have separate nasal ports or simple nasal housing.
Advantages: Commercially available, comfortable.
Disadvantages: Difficult to fabricate for some face shapes; heavy; the putty hardens and requires replacement every 2-4 months; relatively expensive.

Interface: Interface for Ventilator Assisted Living (IVAL, Interface for Ventilator Assisted Living, available through the Department of Physical Medicine and Rehabilitation at the New Jersey Medical School, Newark NJ)
Description: Custom-molded, acrylic butterfly-shaped, low profile interface covering the nostrils and extending over the cheeks with a soft inner gasket around the nasal opening.[38]
Advantages: Comfortable, lasts 2 years or longer, leak-free air delivery for most patients.
Disadvantages: Must be custom made from a facial impression in supine position for best fit; relatively expensive; gasket material needs to be replaced every 3-9 months.

IPPV via Oronasal Interfaces (ONI)

Interface: Commercially Available Strap-Retained ONI (currently used only in the acute setting)
Description: Modified anaesthesia masks covering nose and mouth.
Advantages: May provide better seal than a nasal or simple mouthpiece interface for the obtunded patient.
Disadvantages: Not shown to be more effective than lipseal or nasal interfaces; risk of aspiration in the event of vomiting; uncomfortable.

Interface: Custom-Molded Strapless ONI (Strap-Less ONI available through the Department of Physical Medicine and Rehabilitation at the New Jersey Medical School, Newark NJ, Fig. 1, and at Dallas Rehabilitation Institute, Dallas TX)[38]
Description: Acrylic bite plate for retention attached to an acrylic extraoral shell. It is open to both nose and mouth, permitting concurrent nasal and mouthpiece IPPV.
Advantages: It is easily removed by tongue thrust; speech is adequate during use; an essentially airtight system.
Disadvantages: Expensive to fabricate; dentition must be adequate and stable; may stimulate excessive oral secretions or gag reflex in some; may initially require frequent adjustments to the shell and bite plate to eliminate leak and optimize comfort.

Bach Discussion

Fletcher: When do you go to noninvasive nocturnal ventilation in a patient with COPD who desaturates when you take away the oxygen?

Bach: In my experience, transition from 24-hour tracheostomy IPPV to noninvasive IPPV is almost solely for the neuromuscular, spinal cord, or polio patient. I have a ventilator unit at Kessler Institute for Rehabilitation, and we admit patients there with COPD. I do switch some of these patients to nocturnal nasal ventilation, sometimes BiPAP, but these have not been 24-hour ventilator users, and I'm not sure how much I'm really helping them. Our experience with two advanced lung disease patients was that they had fewer hospitalizations and complications when they used noninvasive IPPV 24 hours/day. For COPD patients in general, my bias is that if they're retaining CO_2, nasal IPPV might be beneficial to them, but I'd like to see improvements in their nocturnal saturations. Without documented improvements in nocturnal saturation, the argument for resting their muscles is not sufficient for me to use it. I use an Ohmeda oximeter because it provides mean saturations every hour, and the percentage of time the saturation is less than 90%, 85%, 80%, and 70%. That can give me objective evidence that I'm doing the patient some good. I differ from Patrick (Leger) in that I rarely use arterial blood gases on neuromuscular patients, and you don't need to use polysomnography. If you know the patient's vital capacity, and you know the patient is hypercapnic (by capnography) or has desaturations signalling the presence of hypoventilation, you know what you need to do. Polysomnography is useful for the symptomatic patient who has a high supine vital capacity—and you're not quite sure what's going on. Even for that patient, however, once hypercapnia and desaturations are observed, it could be argued that the treatment's the same anyway, so why bother. I'm talking about, for example, myotonic dystrophy or certain of the non-Duchenne myopathies where you may have a 2-liter vital capacity and "sawtooth" desaturations overnight. The patient may or may not be hypercapnic. That kind of patient needs a closer look. But for the routine Duchenne patients, you'd rarely, if ever, have to use blood gas analysis or polysomnography.

Leger: I have a comment about indications. I made a remark yesterday about the long-term complications with tracheostomy. I think that a change to noninvasive ventilation might be indicated in many of these patients because many of these patients were begun on tracheostomy ventilation before nasal ventilation was well-practiced. These patients were perfectly adapted, with good quality of ventilation and no problems with the trach for many years. However, as I mentioned yesterday, the chest disorders evolve in this kind of patient—such as an increase in scoliosis, which in turn causes changes in the relationship between the trachea and the tube. The result can be serious difficulties such as granulomas, pain, and bleeding. Such patients, if they are stable, can be transferred from invasive to noninvasive ventilation.

Bach: The problem with kyphoscoliotic (idiopathic or Pott's disease) or COPD patients is that when they feel better, they don't want to use the ventilator because they're mobile. They can walk around. These patients, as Patrick (Leger) says, do evolve. Many do need aid during the daytime. It is important to maintain alveolar ventilation as normal as possible around the clock. Because if you take a patient who's been underventilating for many years, and you just give him nocturnal nasal ventilation or if you give him oxygen and nasal ventilation, he is going to leak most of the delivered volumes out of the mouth. It's been reported that the patients with hypoxia and little or no CO_2 retention don't do as well with nasal ventilation in the acute setting.[1] It's because the chemotactic response to hypercapnia is dulled and because desaturations have been eliminated by oxygen therapy, and reflex activity does not occur to prevent excessive oral leak. There's no doubt about that. Leger, Dominique, Robert, and I have submitted a paper about this to the *American Review*. When the patient desaturates, he often arouses for a few seconds, normalizes his ventilation by cutting off the leak, and then desaturates again. But most patients remain clinically well despite these desaturations. Our patients who have used negative-pressure body ventilators for over 30 years have had terrible desaturations, but the great majority do not have symptoms or any clinical difficulties. So, you can disrupt sleep more than you think, and the patients do fine. Now I'm hypothesizing that for my patients who use mouthpiece-IPPV with a lipseal, for whom ventilation is passive since oral leak is prevented by the lipseal, sleep is probably much better than in kyphoscoliotics using nasal ventilation who are leaking out of their mouths. Although the kyphoscoliotic nocturnal nasal IPPV users have improvements in nocturnal blood gases and do well without a trach, problems are going to arise when they need aid during the daytime. The same thing would be true for any of these restrictive patients.

REFERENCE

1. Wysocki M, Tric L, Wolff MA, Gertner J, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure. Chest 1993;103(3):907-913.

Celli: I want to address Rick's (Fletcher) question. The data from the controlled studies that I know of (the biggest one was the Montreal study) on negative pressure ventilation in COPD showed that there was no indication for noninvasive negative pressure ventilation whether they had CO_2 retention or EMG abnormality.[1,2] The Montreal group had 190 patients with a control group, including a sham. To me that remains an issue of research in COPD population. The only data that I know on nasal positive pressure ventilation is from Nick Hill's group,[3] which Patrick presented, in a randomized trial, again, showed that out of 26 patients only 7 were able to adhere to the technique. When you looked at them, there was no difference in any of the outcomes except some in psychologic testing at the end. So, even though the occasional patient, whom we have all seen—the COPDer with a CO_2 of 110 mm Hg, didn't want to be tubed, we put him on a pressure-limited ventilator and he got better, the literature suggests that we shouldn't be walking around putting COPD patients on ventilators unless we're sure that we know how to handle them.

REFERENCES

1. Martin J, Levy R. Respiratory muscle rest. In: Tobin M, ed. Respiratory muscles. Philadelphia: JB Lippincott, 1990:534.
2. Celli B, Lee H, Criner G, Bermudez M, Rassulo J, Gilmartin M, et al. Controlled trial of external negative ventilation in patients with severe chronic airflow obstruction. Am Rev Respir Dis 1989;140(5):1251-1256.
3. Hill NS, Eveloff SE, Carlisle CC, Goff SG. Efficacy of nocturnal nasal ventilation in patients with restrictive thoracic disease. Am Rev Respir Dis 1992;145(2, Part 1):365-371.

Hill: The need for round-the-clock ventilatory support may be partly related to differences in the patient populations we serve. Very few of my patients receive round-the-clock ventilatory assistance. Most of them are on nocturnal support only; when they get into trouble, they extend the amount of time at night or they might add some time in the afternoon. But I try to preserve some ventilator-free time for them during the daytime, and I permit daytime P_{CO_2}s as high as the upper 40s or maybe the low 50s (torr). These patients have few symptoms and no signs of cor pulmonale. I monitor them nocturnally, and their O_2 saturations remain acceptable overnight. Am I doing something wrong or would you do it differently?

Bach: Are you saying that when your patients need more than, say, 20 hours a day of support, that you then trach them? Of course you want to encourage a patient to have ventilator-free breathing time, but not at the expense of severe hypercapnia. As long as their ventilation is normal when they're on the ventilator (because they're resting their muscles or for whatever reason), they can tolerate longer periods of time off the ventilator breathing on their own.

Hill: When you say that you want to see the ventilation normal, you're talking about *on* the ventilator.

Bach: Well, definitely on the ventilator. The problem is that many patients are assisted but still underventilated nocturnally, have daytime hypercapnia, and nobody realizes that they need daytime aid. A high CO_2 is found on arterial blood gas analysis, and the pulmonologist tells them to "get trached, because nasal ventilation is not effective anymore." It's not that it's not effective anymore, it's that they need daytime aid now because they're weaker. They've got to gradually increase their aid periods. I don't mind them being somewhat hypercapnic when they're breathing on their own but not too severely and not for too long.

Sortor-Leger: I have questions and comments to make about the highly dependent patient who needs, potentially, 23 or 24 hours of ventilation. That is the minority, certainly in our patients, and I agree with you that we try not to do 23-hour ventilation, rather to do 16 or 17 hours of ventilation. We permit them to have a slightly abnormal CO_2 if they tolerate that well, and if it doesn't interfere with their activities of daily living. For those patients who clearly cannot do well without 23 or 24 hours of ventilation, I appreciate your enthusiasm for noninvasive positive pressure or negative pressure ventilation, and I, too, share in that enthusiasm. However, through the years that I have worked with the highly technology-dependent patient, I have learned that it's not always the best system of ventilation. My thinking now is that in some situations we need to be very cautious and careful in sending patients home with noninvasive techniques. In some situations I think it is safer for these patients to have a tracheostomy. This issue needs to be explored, particularly in those patients who go to a community—and there are many, still, in which no one has any idea about how to manage a patient who uses some of these techniques. I think your success clearly indicates that you have a community that is very well trained and well adapted to using these techniques. You have a good system at your university to manage and support these patients, but there are many communities that are not set up that way. We had several experiences that were very tragic. We have sent patients, well ventilated, with very good systems of noninvasive ventilation to many communities that have had no clue how to manage them. Some patients have had no problems but others have and when they had problems, they encountered life-threatening situations because of the lack of knowledge regarding the system they were discharged with. In hindsight it probably would have been better for these patients to be discharged with a tracheostomy. So, I think your enthusiasm is good and if you can create a good support system that's wonderful; but to be realistic, if you don't have those systems, people survive and do very well on trach ventilation, also. Rather than endanger them in an environment that's not well supported, don't be afraid to use tracheostomy. I've changed my attitude over the years about that.

Bach: Yes, but Sue (Sorter-Leger), there are three things. One, if the patient learns how to frog-breathe, and the majority can learn, they're much safer without the trach than with it.

Sortor-Leger: The only study done in regard to that, at Rancho Las Amigas Hospital, showed less than a 50% success rate in learning glossopharyngeal breathing (GPB).[1,2] We have had patients learn GPB spontaneously; of those who were taught, our results are close to what Dr Dail found—leaving many patients unable to use GPB as a backup system.

REFERENCES

1. Dail CW. "Glossopharyngeal breathing" by paralyzed patients: a preliminary report. California Med 1951;75:217-218.
2. Dail CW, Zumwalt M, Adkins H. A manual of instruction for glossopharyngeal breathing. New York: National Foundation for Infantile Paralysis, 1955.

Bach: Well, we've done better than 50%, but that brings to mind another question, irrespective of glossopharyngeal breathing, Is 24-hour aid safer with or without the trach tube? When I'm in doubt, I do what I always say to Bart (Celli). I ask the patient. I surveyed 1,000 predominantly neuromuscular patients around the country who have Lifecare ventilators.[1] I don't know any of these people. I was looking for people who had used both tracheostomy and noninvasive aids. I found 168. Two of them had switched, twice, initially from noninvasive to trach, and then back to noninvasive again. I asked them which they preferred for swallowing, speech, safety, sleep, appearance, and overall. The patients who went from body ventilators to trach were 50-50 in general; they preferred trach for safety and the body ventilators for convenience and comfort. The patients who switched from mouthpiece and/or nasal ventilation to trach overwhelmingly preferred what they had been using before, and many said that they wanted to switch back. Many wrote on the back of the survey that they would go back to noninvasive aid if they could find a physician who would remove the trach tube. Of the patients who went from trach to noninvasive (and these were patients whose average use of ventilatory support was more than 18 hours a day), it was absolutely unanimous. Every one of them said that they liked this much better; the overwhelming majority found it safer, and many wrote that they'd rather be dead than get trached again.[1] So, if you ask the polio, spinal cord, or neuromuscular patient which they prefer and why, you find a very different attitude from that the clinician has regarding safety and desirability. I asked the same patient population of 621 ventilator users (I haven't completed this part of the study yet) to list when they were hospitalized and when they've had pneumonias or severe pulmonary complications. In this population, some have used both trach and noninvasive aids, some have used only trach IPPV, and some have used just noninvasive aids. I also asked them how many hours a day they were using aids. The population that had the least number of hospitalizations and complications were the people who used noninvasive ventilation greater than 16 hours a day. It was significantly less than for patients who used noninvasive ventilation only overnight and significantly less than for patients who used trach ventilation for any daily periods. In every hospital that I've worked in, I've seen patients die because they were accidentally disconnected. Nurses say the low pressure alarm doesn't go off because the hose-trach adapter rests against the skin and no one is there to hook it back up. So, it goes both ways. I believe that for the paralytic/restrictive patient (provided that bulbar musculature is adequate), the noninvasive systems are safer than using tracheostomy IPPV. This is especially true when the patient can learn glossopharyngeal breathing.

REFERENCE

1. Bach JR. A comparison of long-term ventilatory support alternatives from the perspective of the patient and care giver. Chest 1993;104(6):1702-1706.

Use of Negative Pressure Ventilation, Rocking Beds, and Pneumobelts

Nicholas S Hill MD

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Introduction

Introduced during the poliomyelitis, or 'polio,' epidemics of the late 1920s and 1930s,[1] the tank type of negative pressure ventilator was the first mechanical ventilator to see widespread use. The rocking bed, introduced during the late 1940s, was also used widely during the polio epidemics.[2] The Pneumobelt, developed when the polio epidemics were drawing to a close,[3] has seen sporadic use. These so-called body ventilators were largely supplanted during the late 1960s and 1970s by invasive positive pressure ventilators, although they enjoyed a brief resurgence during the early 1980s when it was recognized that they were effective in providing intermittent nocturnal ventilatory assistance to patients with chronic respiratory failure due to neuromuscular disease or chest-wall deformities.[4-6] However, the increasing use of noninvasive positive pressure ventilators over the past 6 years has once again relegated body ventilators largely to the confines of the storeroom. Despite their current status as second-line ventilators, they still have potential important clinical applications, and a knowledge of their function and uses is still desirable for the respiratory care practitioner. In this paper, I review the history, types of equipment available, mechanisms of action, and current clinical applications of each of the devices.

Equipment

Negative Pressure Ventilators

In an historical review, Woollam[7] traced negative pressure ventilation as far back as 1843, when Dalziel published a description of a tank ventilator. However, commercial development of negative pressure ventilators awaited the availability of a dependable electrical power source and the demand

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for ventilatory support of a large population of patients with respiratory failure as occurred during the polio epidemics. The first widely used, commercially available tank ventilator was developed in the late 1920s by Drinker, an engineer at Harvard University.[1] Subsequent modifications were made by the JH Emerson Company, Cambridge MA, leading to an unsuccessful lawsuit by Drinker for patent infringement. The Emerson iron lung (Fig. 1) gained wide acceptance and remains the most commonly available tank ventilator in the United States today. The prototype tank ventilator consists of a rigid cylinder surrounding the patient's entire body, with only the head protruding through a neck seal. The principle of operation is the same as that of all negative pressure ventilators: A subatmospheric pressure is generated around the thorax and abdomen, increasing transpulmonary pressure and causing atmospheric pressure at the mouth to inflate the lungs. With return of intrachamber pressure to atmospheric (or supra-atmospheric) levels, elastic recoil of the lungs and chest wall brings about expiration.

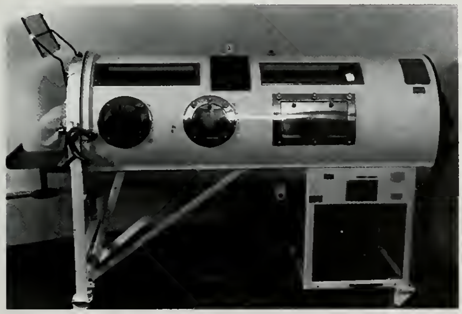

Fig. 1. Emerson iron lung. The user's head protrudes through the neck seal on the left. Excursions of a pulley-driven leather bellows at the opposite end generates pressure swings within. (Reprinted from Reference 6, with permission.)

The chief advantage of the tank ventilator is that it is very efficient and reliable, applying negative pressure to the entire thorax and abdomen and minimizing air leakage by requiring only a neck collar. Therefore, it is capable of supporting patients with severe respiratory muscle weakness or during acute exacerbations of respiratory failure. However, it is large (length 3 m) and heavy (weight 300 kg), virtually precluding portability. It is also very confining, and claustrophobic reactions are common. The patient lies on a relatively comfortable mattress, but the need to remain more or less supine promotes musculoskeletal back and shoulder pain. Also, nursing care and physical therapy are restricted, despite the portholes on the sides that are meant to facilitate access to the patient. Modifications of the tank ventilator include "alligator" or "bi-valve" types[8] that allow a hatch over the patient to be opened, facilitating access, and a fiberglass version (Portalung, Lifecare Inc, Lafayette CO) that weighs 50 kg, enhancing portability.[9]

Other versions of negative pressure ventilators address some of the limitations of tank ventilators. The wrap ventilator (Numowrap, Lifecare Inc), introduced during the 1950s,[10] consists of an airtight jacket that wraps around a rigid semicylindrical chest piece that is positioned over the anterior thorax and abdomen (Fig. 2). The jacket is sealed at the arms, hips, and neck and is connected to a negative pressure generator that produces intermittent subatmospheric pressure within it. The chest piece prevents collapse of the jacket onto the chest wall and abdomen. A number of different jacket styles are available, including plastic ponchos and Gortex body suits. The latter are more efficient than ponchos because they have pantlegs that prevent sucking of the suit under the chest piece. Greater portability is the main advantage of the wrap over the tank ventilator, but I have found it to be less efficient, often causing musculoskeletal back and shoulder pain, and cumbersome to apply, particular-

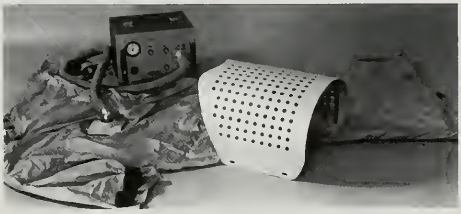

Fig. 2. 'Wrap' ventilator. Nylon poncho is shown connected to Thompson Maxivent negative pressure generator (Puritan-Bennett Inc) in background. Right arm sleeve with Velcro tightening strap is in foreground. At right is rigid plastic cage that fits over chest and abdomen.

ly in quadriplegic patients. Maintaining a tight air seal can be difficult, and patients with severe chest-wall deformities may not fit comfortably underneath the chest piece.

The cuirass or tortoise shell is another variety of negative pressure ventilator that was first introduced during the early 1900s.[7] It consists of a rigid dome that is fitted over the anterior chest wall and abdomen (Fig. 3). It is the least efficient of the negative pressure ventilators because it applies negative pressure to the smallest area. Early studies demonstrated that efficiency was improved if it was placed over the chest and abdomen rather than the chest alone.[11] The shell ventilator is highly portable and, once properly fitted, is easily applied. However, fitting is difficult in patients with chest-wall deformities, and chest discomfort and abrasions are common even after custom-fitting. Also, because of relative inefficiency, the shell and the wrap ventilator are not well suited for management of acute respiratory crises.

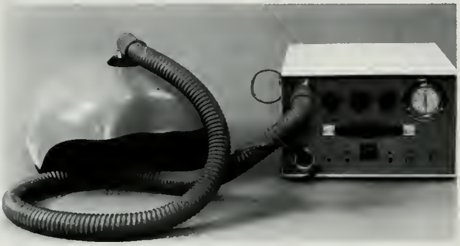

Fig. 3. Cuirass or 'shell' ventilator. The rigid dome on the left fits over the chest and abdomen and is shown connected to a Monaghan 170C negative pressure generator (Lifecare Inc).

A number of negative pressure generators may be used to power the wrap or shell ventilator. These include models from JH Emerson Inc, the Monaghan 170C (Lifecare Inc, Lafayette CO), Thompson Maxivent (Puritan-Bennett, Lenexa KS), and the NEV-100 (Lifecare Inc). These ventilators weigh 10-25 kg and are less portable than some pressure-limited positive pressure ventilators currently available. Also, patient-triggering modes are not available, except with the NEV-100. Negative pressure devices are available by rental through Lifecare Inc, either directly to the patient or through a home respiratory services vendor.

Abdominal Displacement Ventilators

Both the rocking bed and intermittent abdominal pressure respirator (eg, Pneumobelt) rely on displacement of the abdominal contents to effect diaphragmatic motion. Eve[12] first described the technique of manual rocking during the early 1930s. The patient was placed supine on a stretcher and rocked up and down approximately 45° on a fulcrum placed at waist level. The "weight of the viscera pushed the flaccid diaphragm alternatively up and down," achieving artificial respiration. The technique was subsequently adopted by the British Navy as the recommended means of resuscitation for drowning victims.[13] The introduction of rocking beds as ventilatory aids awaited the late 1940s, when Wright described the management of respiratory insufficiency using an oscillating bed that had originally been designed to assist circulation.[14] This led to the development of the McKesson Respiraid Rocking Bed in 1950,[15] intended mainly as an aid to weaning patients with poliomyelitis from dependence on a tank respirator.[16] It facilitated nursing care and enhanced patient freedom but was noisy, bulky, and heavy (455 kg).

The Emerson Rocking Bed (JH Emerson Co, Cambridge MA), also introduced about 1950, was quieter and lighter than the McKesson bed and became the dominant model. Hundreds of rocking beds were manufactured between 1950 and 1960 (JH Emerson, personal communication, 1993), but demand fell drastically after development of an effective vaccine and consequent control of the polio epidemics. Many survivors of the polio epidemics continued to use rocking beds for ventilatory support, sometimes for decades,[17] but most have since expired or switched to other ventilators, and present-day use is limited. Lifecare Inc, a major supplier of rocking beds, estimates that it currently rents 80 rocking beds to users throughout the United States (J Sealy, personal communication, 1993.)

The action of the rocking bed can be compared to that of a piston within a cylinder.[12] As the subject's head moves down, the piston-like viscera and diaphragm slide cephalad within the cylinder-like chest wall, assisting exhalation (Fig. 4). In the foot-down position, the abdominal contents and diaphragm slide caudad, assisting inhalation (Fig. 5). Early studies on the efficacy of rocking showed that

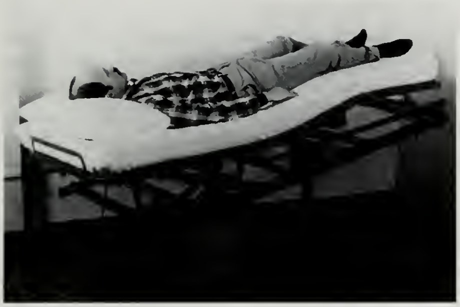

Fig. 4. Rocking bed in head-down position. Abdominal viscera slide cephalad, assisting exhalation. (Reprinted from Reference 6, with permission.)

it produced tidal volumes ranging from a few hundred milliliters up to a liter, and that tidal volumes tended to fall when rocking rate exceeded 16/min.[18] Plum and Whedon[2] found that it was not as effective as the tank respirator, producing adequate alveolar minute volumes in 11 stable patients with respiratory paralysis due to poliomyelitis but not in 5 acutely ill polio patients.

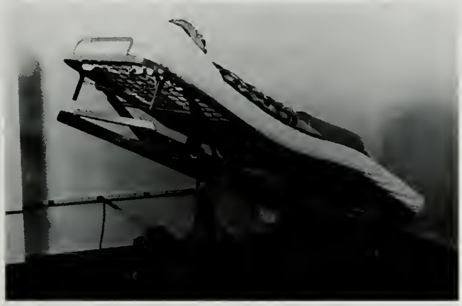

Fig. 5. Rocking bed in head-up position. Viscera slide toward feet, assisting inhalation. (Reprinted from Reference 6, with permission.)

Colville et al[19] subsequently found that the wide individual variations in tidal volumes in response to rocking were related to variations in compliance and effective length of the abdomen. They also found that the greatest displacement of the diaphragm occurred during rocking from the horizontal to the 40° foot-down position (Fig. 6). Likewise, relatively little displacement of the diaphragm occurred during rocking from the horizontal to the head-down position. Joos et al[20] found that tidal volumes were greater in some patients when rocking was achieved entirely in the head-up position (5 to 42° above the horizontal plane) rather than between the head-down and head-up positions (10° below to 27° above the horizontal plane). Taken together, these findings show that the rocking bed is relatively ineffective at high rocking rates or in patients with low abdominal compliances (ie, with severe kyphoscoliosis or excessive thinness or obesity), and is more effective when the rocking is adjusted to keep the head up most of the time, an adjustment that also improves comfort.

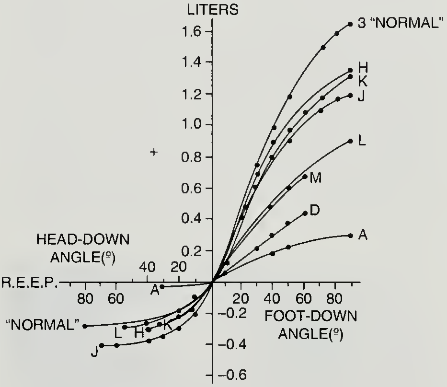

Fig. 6. Relationship between angle of rocking bed tilt (horizontal axis) and lung volume in liters (vertical axis) in 3 normal volunteers and different polio patients as indicated by letters. REEP (resting end-expiratory position) is shown as the angle from horizontal. Note the larger volume changes in the foot-down compared to head-down position, and the wide variations among subjects. (Reprinted from Reference 19, with permission.)

Rocking beds may be rented from Lifecare Inc and are still manufactured by the JH Emerson Co, although only approximately 6 have been made during the past decade (George Emerson, personal communication, 1993). Today's beds are identical to those first introduced during the late 1940s, consisting of a bed frame that is suspended on an axis 100 cm above the ground. They are bulky (193-cm length and 84-cm width) and heavy (approximately 136 kg). Although the axis of rotation can be adjusted, they are usually set to rotate 10° in the head-down direction and 27° foot-down. Adjustable cranks allow raising of the head and knees to minimize slippage; however, excessive flexion of the hips may impair functioning. A foot rest may also be attached to prevent downward sliding, but with proper positioning of the head and knees this is usually unnecessary.

A prototype of the Pneumobelt was used during the late 1930s (Bragg-Paul pulsator),[21] but the currently available version was introduced toward the end of the polio epidemics.[3] It is designed to allow complete freedom of the upper extremities and mouth during use in the sitting position and intended mainly as a daytime ventilatory aid during meals or wheelchair use (Fig. 7). Like the rocking bed, the intermittent abdominal pressure respirator typified by the Pneumobelt (Lifecare Inc, Lafayette CO) has seen only limited use since polio has been controlled. Lifecare estimates that it currently rents 40 Pneumobelts throughout the United States (J Sealy, personal communication, 1993). Several modifications of the abdominal belt respirator have been reported since the original description, including a piston-like device that compresses the abdomen while the patient sits in a wheelchair[23] and a combination of the abdominal belt and intermittent positive pressure breathing; however, these have never gained wide acceptance.[24]

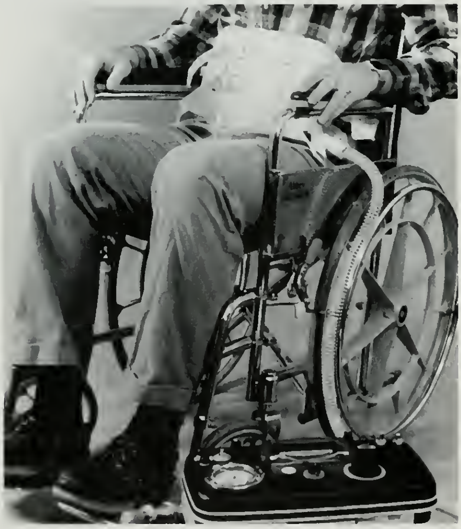

Fig. 7. Pneumobelt is shown, consisting of a rubber bladder (not visible) within the corset held firmly over the anterior abdomen by straps. Positive pressure generator in foreground is a Thompson Bantam (Puritan-Bennett Inc). (Reprinted from Reference 22, with permission.)

The Pneumobelt consists of an inflatable rubber bladder held firmly over the abdomen by an adjustable corset. It operates by a mechanism similar to that of the rocking bed in that it assists diaphragmatic motion by causing piston-like motions of the abdominal viscera within the thoracic 'cylinder.' Inflation of the bladder by a positive pressure ventilator squeezes the abdomen, forcing the diaphragm cephalad and actively assisting exhalation. Upon deflation of the bladder, gravity pulls the viscera and diaphragm back to their original position, assisting inhalation (Fig. 8). Because of this dependence on gravity, the Pneumobelt must be used in a sitting position, optimally at an angle of 45° or greater. At an angle of less than 30°, the ability to assist ventilation is markedly diminished; thus, nocturnal use of the Pneumobelt is limited to patients who can sleep in a sitting position.[25]

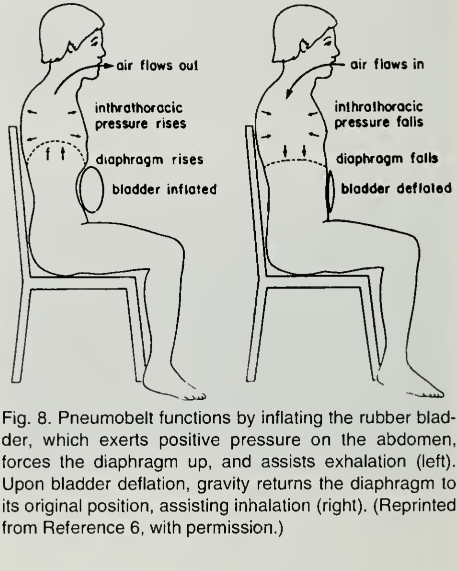

Fig. 8. Pneumobelt functions by inflating the rubber bladder, which exerts positive pressure on the abdomen, forces the diaphragm up, and assists exhalation (left). Upon bladder deflation, gravity returns the diaphragm to its original position, assisting inhalation (right). (Reprinted from Reference 6, with permission.)

The ability of the Pneumobelt to augment tidal volume is linearly related to inflation pressure of the bladder between approximately 15 and 50 cm H_2O.[3]

Pressures exceeding 60 cm H_2O are rarely tolerated because of abdominal discomfort. As with the rocking bed, the ability of the device to augment tidal volume varies considerably among individuals.[3] Important factors that contribute to this variability include abdominal and chest-wall compliances. High abdominal compliance favors efficient functioning of the device, but high chest-wall compliance may allow expansion of the chest wall during bladder inflation, reducing efficiency; thus, as with the rocking bed, body habitus is an important determinant of ventilator efficiency for the Pneumobelt. Although thin patients can be ventilated effectively,[26] both devices work inefficiently in patients with extreme thinness or obesity or severe kyphoscoliosis. The sitting position in patients with severe kyphoscoliosis often brings the lower rib cage in contact with the thighs, precluding proper positioning of the Pneumobelt.

The Pneumobelt is available by rental from Lifecare Inc or through home respiratory services vendors. It can be powered by almost any positive pressure ventilator, but simple, relatively inexpensive pressure-limited ventilators like the Bantam Lightweight (Puritan-Bennett Inc) are most often used.

Clinical Applications

Negative Pressure Ventilators

Because of their reliability, tank ventilators have been used in virtually every form of respiratory failure. If the upper airway was obstructed or swallowing severely impaired, the tank ventilator could be used in combination with a tracheostomy. Early case reports described the use of tank ventilators in treating acute pulmonary edema[27] and pneumonia,[28] and, more recently, reports have been published describing applications in acute exacerbation of chronic obstructive pulmonary disease (COPD).[29,30] In these latter reports, carbon dioxide tension was lowered and tolerance of supplemental oxygen was improved. However, from a pragmatic viewpoint, the application of body ventilators in the treatment of acute respiratory failure is severely limited. Only the tank ventilator is sufficiently efficient to be used in this circumstance, and because it provides no direct access to the airway, problems with secretion removal and atelectasis commonly occur in patients with increased airway secretions. If an endotracheal tube is placed, positive pressure ventilation is a much more convenient means of providing ventilatory support. Occasional patients who have been using body ventilators and who then develop acute respiratory infections may prefer treatment with an iron lung instead of endotracheal intubation, but even these patients require temporary endotracheal intubation if secretions become copious. The tank ventilator may also be used to facilitate extubation following bouts of acute respiratory failure, but, once again, positive pressure ventilation administered noninvasively has largely supplanted this application.

The major application of negative pressure ventilation has been in the treatment of patients with chronic respiratory failure.[8] Much experience with this application was gained during the polio epidemics, but more recent reports have confirmed the earlier experience. During the early 1980s, Garay et al,[4] in patients with a variety of neuromuscular and chest-wall diseases, and Curran,[5] in patients with Duchenne muscular dystrophy, reported sustained improvements in gas exchange and symptoms of hypoventilation after months to years of nocturnal negative pressure ventilation. Splaingard et al[31] subsequently reported 40 patients with a variety of end-stage chest-wall diseases treated with tank ventilators for an average of 11 years with a survival rate of 61%. More recently, Kinnear et al[32] demonstrated that cuirass ventilation in 25 patients with chronic respiratory failure due to kyphoscoliosis, thoracoplasties, or neuromuscular disease had improved carbon dioxide tensions after 1 year of nocturnal ventilatory assistance. In a separate report,[33] these authors also demonstrated that the cuirass produced improvements in minute volume related to increases in peak negative pressure and frequency of the ventilator. Prospective, controlled trials have not been done, largely for ethical reasons, but the aforementioned studies strongly support the contention that negative pressure ventilation in patients with chest-wall disease or central hypoventilation is efficacious in improving gas exchange and symptoms and, most likely, survival.

Recent attention has also focused on the use of negative pressure ventilation in patients with chronic respiratory failure due to COPD.[34] This interest stems from earlier reports showing that negative pressure ventilation effectively rests respiratory muscles in patients with COPD,[35] and that respiratory muscle strength improves after brief periods of assisted ventilation using negative pressure ventilators, presumably due to resting of chronically fatigued muscles.[36,37] A few long-term studies, lasting months, also demonstrated sustained improvements in gas exchange and muscle strength in patients with COPD.[38,39] However, these studies were uncontrolled, and improvements may have been related to other aspects of the rehabilitation program. Subsequently, several longer-term controlled trials have shown no benefit of wrap ventilation over standard therapy in stable COPD patients.[40-42] In the largest study, Shapiro et al[42] randomized 184 patients with severe COPD to wrap or sham ventilation, and even though they demonstrated respiratory muscle rest, they could show no long-term improvements in gas exchange or respiratory muscle strength. Tolerance of negative pressure ventilation was poor in these latter studies, and daily duration of use was less than recommended by the investigators, possibly contributing to the negative results. Nevertheless, these latter results have called into question the efficacy of negative pressure ventilation in treating stable severe COPD patients.

Abdominal Displacement Ventilators

Considering that the rocking bed and Pneumobelt share similar mechanisms of action, it is not surprising that they have similar applications. Because both assist ventilation by augmenting diaphragm motion, they are particularly useful in patients with bilateral diaphragmatic weakness or paralysis. Abd et al[43] demonstrated the utility of this application in 13 patients with bilateral diaphragmatic paralysis following open heart surgery. The rocking bed facilitated weaning from conventional positive pressure ventilation in these patients, who continued to use it nocturnally until phrenic nerve function recovered after 4 to 27 months.

The rocking bed and Pneumobelt may also be used in the management of chronic respiratory failure caused by a variety of slowly progressive neuromuscular syndromes that weaken the diaphragm but leave upper airway function intact. These include Duchenne and limb-girdle muscular dystrophies,[44,45] myotonic dystrophy, and the postpolio syndrome. Occasional patients with other neuromuscular syndromes, such as amyotrophic lateral sclerosis or multiple sclerosis, may also be appropriate candidates if progression of their disease is sufficiently slow and upper airway function has been spared. The Pneumobelt has also been used in patients with high spinal cord lesions, mainly as a daytime supplement, often in combination with nocturnal positive pressure ventilation administered noninvasively or via a tracheostomy.[25,45] In this setting, patients prefer the Pneumobelt because it can be hidden under clothing and does not encumber the face or hands. It fits easily on a wheelchair so that mobility is preserved, and it may be used for around-the-clock ventilatory support in selected patients with high cord lesions. Among these patients, those with the least ability to sustain spontaneous breathing prefer the Pneumobelt over positive pressure systems.[45]

Because both ventilators have limitations that are influenced heavily by the patient's body habitus, a number of caveats should be borne in mind. Neither ventilator is suitable for use in acute respiratory failure because a period of adaptation is necessary for optimal efficiency, and excessive secretions interfere with function. In addition, patients should have an appropriate body habitus, adequate upper airway function, and a condition that is stable or only slowly progressive so that the anticipated duration of use will justify the effort and time required for adaptation. Because the rocking bed is best suited for nocturnal use and the Pneumobelt for daytime use, a patient might use the rocking bed during sleep and the Pneumobelt for daytime desk work or wheelchair use.

Few studies have compared efficacy of the rocking bed and Pneumobelt with that of other noninvasive ventilators. In acutely anesthetized intubated subjects, Bryce-Smith and Davis[18] showed that the rocking bed produced barely adequate tidal volumes when rocking through an arc of 40° and was much less effective than negative pressure ventilators, including the tank ventilator or chest cuirass. More recently, Goldstein et al[46] demonstrated that cuirass-type negative pressure ventilators more effectively augmented tidal volumes and suppressed diaphragmatic electromyographic activity than did the rocking bed in 7 patients with neuromuscular disease. This was a short-term daytime study, and the rocking bed may have been more effective at reducing diaphragmatic electrical activity had patients been monitored overnight after a suitable adaptation period. Even so, the conclusion seems justified that the rocking bed is usually less effective than negative pressure ventilators.

Body Ventilators vs Noninvasive Positive Pressure Ventilation

Many patients with chronic respiratory failure could be managed successfully with one or more of a variety of ventilator options. Thus, it is important for the respiratory care practitioner to appreciate the relative advantages and disadvantages of the options in order to assist the patient in making an appropriate selection. In recent years, noninvasive positive pressure systems have gained popularity because of a number of advantages over body ventilators. Consisting of an interface that weighs ounces and is held in place by straps that can be applied within minutes, positive pressure systems are more convenient to use than most body ventilators. The tank ventilator and rocking bed require lifting the patient onto a mattress, and the wrap ventilator is time-consuming and cumbersome to apply in severely weakened or paralyzed patients. Positive pressure systems are also more portable than body ventilators, with very compact positive pressure ventilators weighing as little as 5 kg (BiPAP System, Respironics Inc, Murrysville PA). Comfort is difficult to compare among the various systems because it depends so much on individual preference. Positive pressure systems encumber the body less than do body ventilators, but some patients cannot tolerate foreign objects on the face. Overall, the rocking bed is probably the most comfortable of the noninvasive ventilators because it consists only of a mattress without other encumbrances, but other disadvantages limit its use. Cost considerations favor the body ventilators such as the iron lung and rocking bed that have low monthly rental fees; however, because these are usually billed by home respiratory care vendors under standard ventilator codes, cost differences are not usually a determining factor.

The considerations previously discussed are important, but two other considerations are overriding because they relate to safety. The first is effectiveness, which depends on individual factors such as body habitus, severity of underlying lung dysfunction, and degree of chest-wall deformity. As discussed previously, patients with severe kyphoscoliosis, extreme thinness, or obesity are ventilated ineffectively by the Pneumobelt and rocking bed, and only slightly more effectively by negative pressure ventilators. Thus, noninvasive positive pressure ventilation is preferred for these patients.

The second consideration is the frequent occurrence of obstructive sleep apneas during nocturnal use of body ventilators. This propensity was first noted in 1951 among normal subjects hyperventilated in a tank ventilator.[47] More recent studies have demonstrated that wrap ventilators induce obstructive sleep apneas in normal subjects,[48] and that severe oxygen desaturations occur frequently among patients with neuromuscular disease receiving negative pressure ventilation. Our group observed that 9 of 12 patients with Duchenne muscular dystrophy using tank or wrap ventilators had nocturnal oxygen desaturations to below 85%. Among 31 patients with a variety of neuromuscular diseases, Bach et al[50] noted significant oxygen desaturations in at least 50%. An underlying sleep apnea disorder is common among patients with severe neuromuscular disease[51] and undoubtedly contributes to the occurrence of these events in some patients during negative pressure ventilation. However, negative pressure ventilation appears to aggravate the condition.[52] Ventilator-triggered breaths may precede the pre-inspiratory activation of upper airway muscles that occurs during normal breathing, rendering upper airway structures collapsible as airway pressure is lowered.[50] Recently, a patient-triggered negative pressure ventilator (NEV-100, Lifecare Inc) has become available, but its effect on induction of obstructive sleep apneas has not been assessed.

Noninvasive positive pressure ventilation ameliorates the obstructive apneas and oxygen desaturations that occur in patients using negative pressure ventilators,[53] and is now considered the preferred mode in most circumstances in which noninvasive ventilation is indicated.[34] Exceptions include patients with facial trauma or burns who cannot use facial interfaces; those with a strong aversion to face, mouth, or nasal interfaces; and others who fail to tolerate noninvasive positive pressure ventilation after concerted attempts with different interfaces.

Indications for Body Ventilators

Because body ventilators are now considered second-line choices for administration of noninvasive ventilation, their use is not indicated unless the patient is unable to use or has failed trials of noninvasive positive pressure ventilation. If this is the case, indications for body ventilators are otherwise the same as for noninvasive ventilation in general (Table 1). The major indication is symptomatic chronic respiratory insufficiency or failure. In most cases, P_{aCO_2} is elevated (> 45 torr) and symptoms include morning headaches, hypersomnolence, and easy fatiguability. Occasionally, nightmares or enuresis are present. In addition, patients should have an appropriate diagnosis such as stable or slowly progressive neuromuscular disease, severe chest-wall deformity, or central hypoventilation. Indications in patients with chronic respiratory insufficiency due to COPD are unclear. Patients should have adequate upper airway function and no excessive secretions. For patients with acute respiratory insufficiency, efficient and reliable body ventilators such as the iron lung can be used if the patient is otherwise stable and has adequate upper airway function. This approach has been used in patients with acute exacerbations of COPD or who have already been using body ventilators for chronic respiratory insufficiency.[29,30]

Table 1. Indications for Body Ventilators*

Symptomatic chronic respiratory failure
  $P_{aCO_2} > 45$ with compensated pH, morning headaches, and hypersomnolence

Appropriate diagnosis
- Slowly progressive neuromuscular disease
  - muscular dystrophy
  - postpolio syndrome
  - high cord lesion
  - multiple sclerosis
- Chest-wall deformity
  - thoracoplasty
  - kyphoscoliosis
- Central hypoventilation

Adequate upper airway function

Obstructive sleep apnea excluded

Inability to use or tolerate noninvasive positive pressure ventilation

*Example of body ventilators are iron lung, Pneumobelt, rocking bed, wrap, and cuirass.

Selection Considerations

Selection of an appropriate body ventilator depends on a number of considerations (Table 2). Patient preferences are, of course, major determinants. If simplicity of operation and freedom of the hands and face are priorities, the rocking bed might be considered in patients who are otherwise candidates. The need for portability favors the wrap, cuirass, or Pneumobelt. Claustrophobic patients should avoid tank ventilators. Efficacy and reliability are also important considerations and may override convenience considerations. If the patient has severe respiratory impairment with little capacity for spontaneous breathing, a reliable and effective ventilator such as the iron lung might be considered. Less efficient ventilators may ventilate such patients adequately, but their efficacy for a particular patient must be demonstrated before they are selected. Chest-wall deformity and body habitus also influence selection because severe kyphoscoliosis and obesity preclude use of the rocking bed and Pneumobelt. In addition, diagnosis may be important; as discussed above, patients with bilateral diaphragmatic paralysis may do well using the rocking bed nocturnally and, if necessary, these patients

Table 2. Selection Considerations for Body Ventilators

Considerations	Possible Selections
Patient preference	
Portability	Wrap, cuirass, pneumobelt
Convenience	Rocking bed, cuirass
Freedom of hands and face	Rocking bed, pneumobelt
Efficiency, reliability	Iron lung
Diagnosis	
Bilateral diaphragm paralysis	Rocking bed, pneumobelt
High cord lesion	Pneumobelt
Obstructive sleep apnea	Positive pressure ventilation, noninvasive or via tracheostomy
Body habitus	
Marked kyphoscoliosis or obesity	Positive pressure ventilation, iron lung

and those with high cord lesions may be adequately supported using a Pneumobelt during the daytime.

Initiation of Body Ventilation

When patients meet criteria for noninvasive ventilation, they are usually briefly hospitalized for initiation of ventilation, although stable patients who are comfortable with new devices may begin as outpatients. The various ventilator options are reviewed with the patient who is encouraged to indicate preferences. Patients who express reluctance about having devices strapped to the face might try certain body ventilators initially, but most patients will begin with noninvasive positive pressure ventilation. If a trial with body ventilators is contemplated, plans should be made in advance because several days may be necessary to obtain an iron lung or rocking bed.

The initial trial should be carried out in a quiet, relaxed setting during the daytime. The patient should be thoroughly familiarized with the equipment, and the respiratory care practitioner may want to demonstrate application on himself first. The equipment is then applied to the patient, and baseline measurements of respiratory rate and tidal volume are taken using a hand-held spirometer. Patients are often apprehensive when first using the devices (particularly with the iron lung when their head must slide through the neck seal and with the wrap ventilator, which may require considerable time for application), and frequent reassurance is necessary. Adequate fitting may be difficult during initial trials with the shell ventilator, even in patients with only mild chest-wall deformities, and custom-molding may prove necessary if this mode is selected. The rocking bed and Pneumobelt usually engender less apprehension, but comfort and fit should be optimized. With the rocking bed, the head and knees are raised slightly using hand cranks to improve comfort and minimize sliding. Pneumobelts are available in three sizes; the straight upper border should extend to the level of the xiphoid and the curved lower border to the pubis. The straps are then adjusted so that the corset is held firmly against the abdomen without constricting it.

No universally accepted recommendations exist for selection of ventilator settings, so the following guidelines are based on the author's experience and published work of others.[2,3,26,41,43] Once the device is applied, pressure settings are reduced to a minimal level while the rate is adjusted. Respiratory rate selection depends partly on anticipated efficiency of the device, the nature of the respiratory problem, and patient comfort. For example, the highly efficient iron lung is often set at a lower rate than the cuirass, and some studies have found that efficiency of the rocking bed falls if rocking rate exceeds 16/min.[54] Other studies have found that patients with high cord lesions achieve adequate ventilation using the Pneumobelt at rates of 12-14/min,[26] whereas the author's experience in patients with muscular dystrophy suggests that they prefer higher rates. In general, a rate that approximates spontaneous breathing rates, as long as it does not exceed the low 20s, seems to be comfortable for most patients.

Once the rate is selected, then pressure is adjusted to augment tidal volume. Selection of the appropriate negative or positive pressure is again a trial and error process that depends on efficiency of the device, chest-wall and abdominal compliances, patient comfort, and desired augmentation of tidal volume. Appropriate targets for augmentation of tidal volume during the initial trial have not been established, but I aim for a 30-50% increase over baseline. Table 3 lists the pressure ranges commonly used with the various devices. As would be anticipated, negative pressures using the iron lung are substantially lower than those necessary to achieve comparable tidal volumes using the wrap or cuirass ventilators.

During the initial trial, frequent ventilator adjustments are made to optimize tidal and minute volumes and comfort. With the negative pressure ventilators, air leaks compromise efficiency, particularly with the wrap and shell ventilators, and these leaks should be located and corrected as much as possible. The neck seal on the iron lung should be tightened just enough to minimize leaks without making the patient uncomfortable. Comfort on the rocking bed is achieved quite readily, and although the rocking excursion can be increased to augment tidal volume, beds are usually adjusted to rock between the standard 10° head-down and 27° foot-down positions. Motion sickness is rare with the rocking bed,

Table 3. Typical Settings, Relative Efficiency, and Rental Costs of Body and Positive Pressure Ventilators

	Settings			Costs (U.S. dollars/month)	
Ventilator	Rate (Breaths/min)	Pressure (cm H_2O)	Efficiency	Primary Device	Pressure Generator
Negative pressure					
Iron lung	12-24	−10 to −35	+++	378*	—
Porta lung	12-24	−10 to −35	+++	562	380
Wrap	14-28	−15 to −45	++	159	380
Shell	14-28	−15 to −45	+	64	380
Pneumobelt	12-24	+15 to +50	++	50	310
Rocking bed	12-24	40°	+	250*	—
Positive pressure	12-24	+10 to +30	+++	453†	—

*Not including $500 delivery charge.

†Allowable Medicare charge for standard portable volume ventilator (DME code EO 450).

presumably because the rocking occurs in one plane only. With the Pneumobelt, pressure is slowly increased until the desired tidal volume or limit of tolerance for pressure is reached. At pressures up to 50-60 cm H_2O, patients usually tolerate the device well, even during meals. Considerable coaching is often necessary to encourage patients to relax while they are adapting to the devices and to try to synchronize their breathing for optimization of ventilatory assistance.

In addition to tidal and minute volumes, gas exchange should be monitored during the initial trial. This can be achieved by obtaining a blood gas measurement after the first hour or two of use or by using pulse oximetry and end-tidal capnography when the patient has only minimal parenchymal lung disease. In my experience, a drop in P_{aCO_2} or P_{etCO_2} of 5-10 torr is desirable during the initial trial, but this is not essential. Some patients have a gradual reduction in P_{aCO_2} after continued use, even if no substantial drop occurs during the initial trial. Hyperventilation can occur in patients with normal lung and chest-wall mechanics who are using more efficient ventilators such as the iron lung or, occasionally, the Pneumobelt.

Monitoring and Follow-Up

After completion of the initial trial, patients are encouraged to gradually extend periods of use as tolerated. The speed of their adaptation depends on their need for ventilatory assistance and acceptance of the device. Patients requiring only nocturnal ventilatory assistance may require days to months before they are sleeping through the night using the device. Adaptation may be enhanced in patients using negative pressure ventilators who are experiencing musculoskeletal pain by adjusting fit and administering nonsteroidal anti-inflammatory drugs. Patients with greater needs for ventilatory assistance usually adapt more rapidly by necessity, but those patients often fare better with a combination of noninvasive devices. Prolonged use of a single device may cause abrasions or pressure sores, particularly with the cuirass and Pneumobelt, and, although padding and fitting modifications may alleviate the problem, the ability to switch to an alternative ventilator is desirable.

Although equipment can be rented directly from equipment suppliers like Lifecare, use of a vendor that also supplies home respiratory therapy services is advantageous, particularly during the initial adaptation period. Therapists should visit the patient in the home as often as is necessary to check on the patient and make adjustments. Once the patient has adapted successfully, home visits can be made less often, but monthly or bimonthly visits are still desirable. Return visits to the physician's office should be made after the first few weeks of use and every few months thereafter, as long as adaptation proceeds smoothly. At the time of office visits, symptoms should be assessed, and the patient should be examined for evidence of respiratory distress and cor pulmonale. Pulmonary function should be monitored, and gas exchange should be measured using either arterial blood analysis or noninvasive devices as discussed earlier. No optimal level for daytime P_{aCO_2} has been determined, but, in my experience, P_{aCO_2} levels up to the low 50s are acceptable as long as symptoms are well controlled and signs of cor pulmonale are absent. In patients with normal lungs, daytime oxygen supplementation is usually unnecessary if P_{aCO_2} is maintained at this level.

Nocturnal monitoring that includes oximetry, chest-wall movement, and nasal and oral airflow should be performed for evidence of obstructive sleep apneas or nocturnal desaturations. If significant abnormalities occur, the patient should be encouraged to reconsider noninvasive positive pressure ventilation. If patients are unable to switch to noninvasive positive pressure ventilation, low levels of nasal CPAP used in combination with the body ventilator may eliminate the sleep-disordered breathing events.[49] Otherwise, tracheotomy should be considered.

If the daytime P_{aCO_2} cannot be lowered sufficiently or if an increase occurs after prior successful stabilization, nocturnal monitoring should be performed to assess ventilator effectiveness. If frequent obstructive apneas are not found, then increasing ventilator pressure or increasing the duration of use may bring the P_{aCO_2} back into the desired range. In my experience, patients need not make frequent office visits after they have been successfully stabilized, but they should be informed about symptoms of hypoventilation and instructed to contact their physician and respiratory therapist should these recur.

Costs vary widely among individual devices. Table 3 gives monthly rental rates of ventilators obtained directly from Lifecare Inc. Devices like the Numowrap and Pneumobelt are inexpensive to rent, but the pressure generator that must be used to power them makes total costs comparable to other devices. Also, although rental rates for the iron lung and rocking bed are relatively low, delivery costs are high. In addition, because the devices are usually obtained from Lifecare via another respiratory home services vendor, Medicare and Medicaid patients are usually billed using standard ventilator codes, and cost differences are thereby eliminated. Nevertheless, body ventilators used in the home are usually less expensive than invasive positive pressure ventilators because tracheostomy supplies are unnecessary.

Conclusions

Body ventilators are considered second-line choices in patients deemed candidates for noninvasive ventilation. However, should these patients be unable to use or adapt to noninvasive positive pressure ventilation, body ventilators should be tried before other alternatives, such as tracheotomy, are considered. Body ventilators can be effective and are generally better tolerated, more easily administered, and less costly than positive pressure ventilation administered via a tracheostomy. To select the body ventilator that is most appropriate for a given patient, patient preference, ventilator efficiency, underlying diagnosis, and body habitus should be considered. In appropriate patients, use of body ventilators can successfully support ventilation for years, even in patients with minimal respiratory muscle function, but these patients require close follow-up both in the home and in the office, and this should include periodic nocturnal monitoring to exclude obstructive sleep apnea.

REFERENCES

1. Drinker P, Shaw LA. An apparatus for the prolonged administration of artificial respiration: 1. Design for adults and children. J Clin Invest 1929;7:229-247.
2. Plum F, Whedon GD. The rapid-rocking bed: its effect on the ventilation of poliomyelitis patients with respiratory paralysis. N Engl J Med 1951;245:235-241.
3. Adamson JP, Lewis L, Stein JD. Application of abdominal pressure for artificial respiration. JAMA 1959;169:1613-1617.
4. Garay SM, Turino GM, Goldring RM. Sustained reversal of chronic hypercapnia in patients with alveolar hy-

poventilation syndromes: long-term maintenance with noninvasive mechanical ventilation. Am J Med 1981;70: 269-274.

5. Curran FJ. Night ventilation by body respirators for patients in chronic respiratory failure due to late stage Duchenne muscular dystrophy. Arch Phys Med Rehabil 1981;62(6):270-274.
6. Hill NS. Clinical applications of body ventilators. Chest 1986;90(6):897-905.
7. Woollam CHM. The development of apparatus for intermittent negative pressure respiration. (2) 1919-1976, with special reference to the development and uses of cuirass respirators. Anaesthesia 1976;31(5):666-685.
8. Shneerson JM. Assisted ventilation. 5. Non-invasive and domiciliary ventilation: negative pressure techniques. Thorax 1991;46(2):131-135.
9. Weingarten WS. Respiration equipment: advancement over the years, equipment now in use and most recent developments. Rehab Gazette 1978;21:34-36.
10. Spalding JMK, Opie L. Artificial respiration with the Turncliffe breathing jacket. Lancet 1958;1:613-615.
11. Collier CR, Affeldt JR. Ventilatory efficiency of the cuirass respirator in totally paralyzed chronic poliomyelitis patients. J Appl Physiol 1953;6:531-538.
12. Eve FC. Actuation of the inert diaphragm. Lancet 1932; 2:995-997.
13. Eve FC. Resuscitation methods for rescue boats. Br Med J 1945;1:21-22.
14. Wright J. The Respir-aid rocking bed in poliomyelitis. Am J Nurs 1947;47:454-455.
15. Carter HA. McKesson Respiraid rocking bed accepted. JAMA 1950;144:1181.
16. Lewis L, Hirschberg GG, Adamson JP. Respiratory rehabilitation in poliomyelitis. Arch Phys Med Rehabil 1957;38:243-249.
17. Sternburg L, Sternburg D. View from the seesaw. New York: Dodd, Mead & Co, 1986.
18. Bryce-Smith R, Davis HS. Tidal exchange in respirators. Curr Res Anaesth Analg 1954;33:73-85.
19. Colville P, Shugg C, Ferris BG. Effects of body tilting on respiratory mechanics. J Appl Physiol 1956;9:19-24.
20. Joos TH, Dickinson DG, Talner NS, Wilson JL. The rocking bed and head position. N Engl J Med 1956;255: 1089-1090.
21. McSweeney CJ. The Bragg-Paul pulsator in treatment of respiratory paralysis. Br Med J 1938;1:1206-1207.
22. Rondinelli RD, Hill NS. Rehabilitation of the patient with pulmonary disease. In: Delisa JA, ed. Rehabilitation medicine: principles and practice. Philadelphia: JB Lippincott, 1988:704.
23. Janelli HV, Krouskop TA, Canzoneri J, Jameson J. Positive pressure respiratory assist for wheelchair-mobile persons. Arch Phys Med Rehabil 1980;61(3):143-144.
24. Gray FD, Field AS. The use of mechanical assistance in treating cardiopulmonary disease. Am J Med Sci 1959; 288:146-152.
25. Yang GF, Alba A, Lee M, Khan A. Pneumobelt for sleep in the ventilator user: clinical experience. Arch Phys Med Rehabil 1989;70(9):707-711.
26. Miller HJ, Thomas E, Wilmot CB. Pneumobelt use among high quadriplegic population. Arch Phys Med Rehabil 1988;69(5):369-372.
27. Wolfe RM. Use of tank ventilator for relief of cardiac dyspnea. JAMA 1933;101:598.
28. Miller FL, Zerbi-Ortiz A, Elkins JT. Use of the tank ventilator in overwhelming bacterial pneumonia. N Engl J Med 1960;262:1264-1266.
29. Montserrat JM, Martos JA, Alarcon A, Celis R, Plaza V, Picado C. Effect of negative pressure ventilation on arterial blood gas pressures and inspiratory muscle strength during an exacerbation of chronic obstructive lung disease. Thorax 1991;46(1):6-8.
30. Sauret JM, Guitart AC, Rodriguez-Frojdn G, Cornudella R. Intermittent short-term negative pressure ventilation and increased oxygenation in COPD patients with severe hypercapnic respiratory failure. Chest 1991;100(2):455-459.
31. Splaingard ML, Frates RC Jr, Jefferson LS, Rosen CL, Harrison GM. Home negative pressure ventilation: report of 20 years experience in patients with neuromusuclar disease. Arch Phys Med Rehabil 1985;66(4): 239-242.
32. Kinnear W, Hockley S, Harvey J, Shneerson J. The effects of one year of nocturnal cuirass-assisted ventilation in chest wall disease. Eur Respir J 1988;1(3):204-208.
33. Kinnear W, Petch M, Taylor G, Shneerson J. Assisted ventilation using cuirass ventilators. Eur Respir J 1988; 1(3):198-203.
34. Hill NS. Noninvasive ventilation: does it work, for whom, and how? Am Rev Respir Dis 1993;147(4):1050-1055.
35. Rochester DF, Braun NMT, Laine S. Diaphragmatic energy expediture in chronic respiratory failure: the effect of assisted ventilation with body respirators. Am J Med 1977;63(2):223-232.
36. Cropp A, DiMarco AF. Effects of intermittent negative pressure ventilation on respiratory muscle function in patients with severe chronic obstructive pulmonary disease. Am Rev Respir Dis 1987;135(5):1056-1061.
37. Scano G, Gigliotti F, Duranti R, Spinelli A, Gorini M, Schiavina M. Changes in ventilatory muscle function with negative pressure ventilation in patients with severe COPD. Chest 1990;97(2):322-327.
38. Braun NMT, Marino WD. Effect of daily intermittent rest of respiratory muscles in patients with severe chronic airflow limitation (CAL) (abstract). Chest 1984;85: 59S-60S.
39. Gutierrez M, Beroiza T, Contreras G, Diaz O, Cruz E, Moreno R, Lisboa C. Weekly cuirass ventilation improves blood gases and inspiratory muscle strength in patients with chronic air-flow limitation and hypercarbia. Am Rev Respir Dis 1988;138(3):617-623.
40. Zibrak JD, Hill NS, Federman EC, Kwa SL, O'Donnell C. Evaluation of intermittent long-term negative-pressure ventilation in patients with severe chronic obstruc-

tive pulmonary disease. Am Rev Respir Dis 1988; 138(6):1515-1518.

41. Celli B, Lee H, Criner G, Bermudez M, Rassulo J, Gilmartin M, et al. Controlled trial of external negative pressure ventilation in patients with severe airflow obstruction. Am Rev Respir Dis 1989;140(5):1251-1256.
42. Shapiro SH, Ernst P, Gray-Donald K, Martin JG, Wood-Dauphinee S, Beaupre A, et al. Effect of negative pressure ventilation in severe chronic obstructive pulmonary disease. Lancet 1992;340(8833):1425-1429.
43. Abd AG, Braun NMT, Baskin MI, O'Sullivan MM, Alkaitis DA. Diaphragmatic dysfunction after open heart surgery: treatment with a rocking bed. Ann Intern Med 1989;111(11):881-886.
44. Mohr CH, Hill NS. Long-term follow-up of nocturnal ventilatory assistance in patients with respiratory failure due to Duchenne-type muscular dystrophy. Chest 1990;97(1):91-96.
45. Bach JR, Alba AS. Intermittent abdominal pressure ventilator in a regimen of noninvasive ventilatory support. Chest 1991;99(3):630-636.
46. Goldstein RS, Molotiu N, Skrastins R, Long S, Contreras M. Assisting ventilation in respiratory failure by negative pressure ventilation and by rocking bed. Chest 1987;92(3):470-474.
47. Peltier LF. Obstruction apnea in artificially hyperventilated subjects during sleep. J Appl Physiol 1953;5:614-618.
48. Levy RD, Bradley TD, Newman SL, Macklem PT, Martin JG. Negative pressure ventilation: effects on ventilation during sleep in normal subjects. Chest 1989;95(1): 95-99.
49. Hill NS, Redline S, Carskadon MA, Curran FJ, Millman RP. Sleep-disordered breathing in patients with Duchenne muscular dystrophy using negative pressure ventilators. Chest 1992;102(6):1656-1662.
50. Bach JR, Penek J. Obstructive sleep apnea complicating negative-pressure ventilatory support in patients with chronic paralytic/restrictive ventilatory dysfunction. Chest 1991;99(6):1386-1393.
51. Smith PEM, Calverley PMA, Edwards RHT. Hypoxemia during sleep in Duchenne muscular dystrophy. Am Rev Respir Dis 1988;137(4):884-888.
52. Scharf SM, Feldman NT, Goldman MD, Haut HZ, Bruce E, Ingram RH Jr. Vocal cord closure: a cause of upper airway obstruction during controlled ventilation. Am Rev Respir Dis 1978;117(2):391-397.
53. Ellis ER, Bye PT, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular disease: positive pressure ventilation through a nose mask. Am Rev Respir Dis 1987;135(1):148-152.
54. Gordon AS, Raymon F, Savode M, Ivy AC. Manual artificial respiration: comparison of effectiveness of various methods on apneic normal adults. JAMA 1950;144: 1447-1452.

Hill Discussion

Bach: I agree with everything you said. It's just that there are indications for body ventilator use. The main thing I use them for, as I mentioned, is when I'm removing tracheostomy tubes in patients with no ventilator-free breathing time during conversion to noninvasive intermittent positive pressure ventilation. Another indication is during extubation of a patient with no ventilator-free time and conversion to noninvasive IPPV to avoid tracheotomy. I then ventilate the patient with negative pressure while teaching him mouthpiece and/or nasal ventilation. So, for the patient who hasn't learned how to use noninvasive IPPV before getting intubated, negative pressure can be a nice bridge to noninvasive IPPV.

Sortor-Leger: First, I'd like to underscore what John said. Questionable extubations are sometimes done, not knowing for sure whether the patient will remain extubated successfully. The iron lung in our practice with neuromuscular patients has allowed us on several occasions to extubate questionable patients safely without needing to reintubate. These patients could then go on to use other less obtrusive means of noninvasive ventilation. Second, you showed a Pneumobelt with the Bantam ventilator, and I would like to just mention a few problems that we have encountered. In our practice, we have mostly used the Pneumobelt for daytime ventilation in quadriplegic patients who have the need to keep their mouth free for activities of daily living. Many of our patients use their mouths for activities such as environmental control, computer work, writing, and painting. One of the difficulties we have had with the Bantam is that the battery consumption is very high, resulting in the need for a large battery for minimum time. These batteries are difficult to mount on the wheelchair, especially an electric wheelchair where there is already very little free space. Our solution has been to change to a portable volume ventilator to power the Pneumobelt, which then allows us to use a much smaller battery for a longer period of time.

Hill: That's a very important consideration. One of my patients who used a Pneumobelt ran into a problem because of that. In 1985, Hurricane Gloria wiped out his electric power, and his battery ran out before he could get to a generator. He was intubated and ended up with a tracheostomy after a series of other disasters, but the battery failure initiated the whole thing. This is a real concern, and an adequate backup power source should be available for all patients who depend on ventilatory assistance.

Sortor-Leger: Third, you said that you had difficulty convincing your patients to change ventilator systems, especially from negative pressure. We have also had these difficulties, especially when a patient has organized activities of daily living around a particular ventilator system. Do you have any suggestions on how to help patients make these sometimes medically necessary decisions?

Hill: Well, send their twin to Disney World. That's very strong motivation.

Kacmarek: Well, you need a very strong motivator. The strongest motivator I have seen for switching from negative to positive pressure ventilation was when one twin using a Pneumowrap for muscular dystrophy was unable to travel to Disney World because the Pneumowrap was too bulky; whereas, her twin using positive pressure was able to go. The next year, both twins were using positive pressure and made the trip.

Sortor-Leger: Negative pressure devices are also not as efficient.

Kacmarek: They're very inefficient unless you have a really cooperative patient. They take time and are work. So, I would be really concerned about recommending negative pressure unless you have an iron lung.

Sortor-Leger: Or a portable.

Kacmarek: Or a portable—but a full body closure with which you're sure you have control over the ventilation process.

Bach: A Pneumowrap can work, but it's awfully difficult because it can take many people and a lot of time to put the patient in it.

Kacmarek: It's difficult to get a seal. You don't always get a good seal.

Hill: And the patient has to have confidence that the portable negative pressure devices will work. Even though the Pneumobelt is potentially a very effective ventilator, if you have someone who is very anxious and not 'working with it,' it may not be effective at all.

Fletcher: How do the nurses accept an iron lung in an MICU?

Kacmarek: They go crazy!

Fletcher: I'll bet.

Sorter-Leger: But they're not used to it.

Kacmarek: Yeah, but realistically.

Fletcher: You can't do it. It's very difficult to take care of the patients.

Kacmarek: We've done it once or twice, and had to fight to continue using it.

Fletcher: I don't understand what the point is in going from one type of ventilation to another in a patient we're trying to wean. What do you gain by using an interim form, a different form of ventilation?

Kacmarek: You've all had the experience of when you've changed a patient from an endotracheal tube to a tracheostomy tube that they have weaned shortly thereafter. You've got a nasal endotracheal tube in place, the same size as the tracheostomy but as soon as you change the device because of the alteration in resistance to flow, etc, the patient weans.

Fletcher: But I thought that was what we have pressure support for.

Kacmarek: Even so, . . .

Fletcher: If you overcome the resistance of the nasaotracheal tube with pressure support, or if the patient's able to support his ventilation on zero CPAP with pressure support for so many hours, he's ready to extubate.

Sortor-Leger: Well, except you don't know about the airway.

Fletcher: What do you mean about the airway?

Sortor-Leger: You may have increased airway resistance caused by the trauma of intubation. For a patient with normal respiratory muscle strength, this may not present a problem, but for a patient with very weak and/or partially paralyzed respiratory muscles this can present a major problem after extubation. Noninvasive ventilatory support could be very important.

Kacmarek: But in the typical ICU it is useful to go from an endotracheal tube to face mask ventilation or nasal mask ventilation. You've had such patients. You didn't know if they were going to make it. So, you took the chance, you extubated them, and 4 or 5 hours later, you reintubated that patient. Instead of doing that, take that patient and before you extubate him, size and fit him, talk to him about what you're going to do with a nasal or a full face mask. Extubate him, and be ready to use a noninvasive technique right away, if there are problems or even if there are not, for a short interim let him get used to the device.

Fletcher: You are talking about pressure- or volume-cycled?

Kacmarek: What I would use is pressure support and CPAP on a volume . . .

Fletcher: cycled ventilator?

Kacmarek: Yeah.

Fletcher: Except that the tube's out, you're using the same system to ventilate them except you've extubated them and put on the mask.

Kacmarek: Exactly. And that's what I would recommend to do rather than a negative pressure device.

Hill: The shift from negative to positive pressure ventilation is something that's happened dramatically over the last several years. We have never used negative pressure ventilators to facilitate weaning in my hospital, anyway, although it has been done by others. The only time we use them now is in patients who get into trouble during chronic use of body ventilators. To get these patients through an acute exacerbation, sometimes we use an iron lung because it is the most efficient body ventilator. Our nurses are initially curious about using the iron lung. But when they get used to it they find it very simple, and I seldom get many complaints about it. I think most complaints are from house officers who don't like drawing blood gases from patients in it. And you can have some pretty interesting problems with arterial lines and I.V. administration during negative pressure cycling in an iron lung.

Celli: For whatever it's worth, the group from Denver in 1977, I think, in the *American Journal of Medicine*,[1] prospectively looked at extubations and intubation in ICUs, and showed that premature extubation, or self-extubation, which occurred in 7%, had no bearing on survival or complications afterwards. So what—all those fears that we have! So what, if you've got to tube them again! With positive proportional assist ventilation and nasal positive pressure ventilation, I don't think we'll have to use the negative pressure devices. My question to Nick (Hill) has to do with physiology. I read a few papers from Brazil in which they looked at atrial naturetic factor (ANF) and showed that even though you may ventilate people via the nose, with positive pressure, and think that physiologically it's the same as negative, there are differences in the way you generate ANF. The reason why I mention it is that when we did our study, the first thing that happened after we hooked the patients up was that 4 hours later the patients wanted to go to the bathroom and urinate. That happens and that has been a major problem for negative pressure ventilation. So, I tried to look at whether this was just in their mind, and then these Brazilians came out with ANF. So, there is more to negative pressure than meets the eye. I would like to have your comments on the physiology of negative pressure.

REFERENCE

1. Zwillich C, Jones B. Complications of intubation in intensive care units. Am J Med 1976;57:181-187.

Hill: Well, Whittenburger et al studied the hemodynamic effects of negative pressure ventilation during the 1950s. They didn't find any difference between negative pressure and positive pressure in terms of effects on venous return and cardiac output. There are also studies that show that ANF goes up in patients on positive pressure ventilation, so I think the release of ANF is more likely related to swings in intrathoracic pressure, causing distension of the atrium. This occurs with either form of ventilation, rather than uniquely with negative pressure.

Fletcher: ANF goes up with sleep apnea and there's a diuresis. When you correct it with nasal CPAP, it returns back to normal. So that would go along with your observation that with negative pressure ventilation ANF goes up and there's an increased diuresis.

Bach: I just wanted to point out that for the most part, Bart (Celli), Bob (Kacmarek), and Rick (Fletcher), you've been talking about patients who you think may be extubated and may breathe on their own, or if they extubate themselves they will probably survive anyway. But the more interesting patient to me is, for example, the high level quad whose lungs aren't that unhealthy, but who can't be weaned and has been intubated for a couple of weeks. It comes to the point where you've got to either trach that patient or do something else. You can get by with negative pressure to ventilate them, and then teach them mouthpiece and nasal ventilation. However, if you extubate a patient who can't breathe, and you try to teach him these techniques without body ventilator support, he's going to desaturate, panic, and maybe need to be reintubated. It's not just a question of the borderline patient, but the patient who really needs to be ventilated.

Make: I'm sitting here trying to put together everything I've heard about what happens when you extubate a patient. We've recommended positive pressure ventilation for everybody who gets extubated and also negative pressure ventilation for everybody who gets extubated. What about upper airway obstruction, the patient whom you just extubated who is likely to have some degree of upper airway obstruction? When do you use positive pressure and when do you use negative pressure ventilation? Are our readers going to understand what we're talking about?

Several Voices in Chorus: No!

Celli: It depends on David Pierson! He's got to write the summary. Whatever he wants is what's going . . .

Hill: That's because we don't know what we're talking about. One thing that Bart (Celli) said is that mortality and morbidity are not greater if you merely reintubate patients who fail after an initial extubation. I think when you're in doubt, that's probably what you should do. I think the pragmatic thing to do when you don't want to reintubate is to try positive pressure noninvasive ventilation, either by face or nasal mask. I don't see any clear advantages to using negative pressure at this point, partly because negative pressure ventilators are less readily available. If I were to use a negative pressure ventilator, the iron lung is the only one I would consider. However, unless you have one sitting in your hospital, they take a day to get and cost $500 to transport. So, it's usually not a pragmatic solution. I think the best application of negative pressure for acute exacerbations is in the patient who has already been using one at home. Additionally, if you've tried positive pressure ventilation, and the patient says, "You know, I just hate things on my face. I won't use any kind of positive pressure device." Then it's reasonable to try negative pressure ventilation.

Make: Are there data to indicate which patients we need to extubate carefully and which we need to extubate and immediately ventilate with some other device? Does extubation with the addition of some other form of ventilatory support other than the tube and the ventilator improve outcome?

Hill: That's a very pertinent question. And, questions for future research.

Pierson: Would it be fair to say that, based on current knowledge and everything that's been presented here, positive pressure ventilation should be the first thing considered and that negative pressure ventilation is a possible alternative if there are reasons not to use positive pressure ventilation? Could we make that statement?

Chorus: Yes!

Pierson: Now, we're talking about two distinctly different settings, and I think that has been the source of some of the chafing here, in that extubation as part of a traditional weaning program in the patient who has had acute respiratory failure is a different setting from what John (Bach) has been talking about, that is how to manage indefinitely the patient who, as far as you know, is in a stable permanent state. I think we need to make that distinction clear.

Hill: I see three different settings: the acute, the weaning from the acute, and then the chronic, stable.

Pierson: Right: the acute exacerbation, de novo, managed with only noninvasive; the patient who has been routinely managed with endotracheal intubation and positive pressure ventilation in the ICU who is then subjected to noninvasive ventilation as a transitional measure to ease the discontinuation; and the long-term. Is there a consensus, then, that in 1993, positive pressure ventilation by a nasal mask, mouthpiece, or face mask should be the first mode tried in all of these?

Chorus: Yes, yes.

Pierson: We have in the room several virtuosos, but we're talking about high school music lessons here. There is unquestionable expertise in this room that is unmatched anywhere else in the world, and that expertise is not very relevant to the readers of our journal, who are trying to deal with this issue the first time they encounter it, or the 10th time they've encountered it. So, perhaps with that mild caveat, although there are exceptions in circumstances of unusual individual expertise, by and large in patient management, noninvasive positive pressure ventilation is today the approach of first choice.

Make: If we're going to make a recommendation, I would eliminate the face mask because there aren't currently any readily available face masks suitable for continuous ventilation. You have to think about what equipment is available, and the most commonly available item for people across the country who probably have access to a sleep lab is a nasal mask. If a full face mask is available, it's likely to be an old anesthesia mask. They certainly don't have access to mouthpieces. So, if you're going to start making this recommendation more generalizable, I would consider these points.

Hardy: Two things, the comment about consensus—I think I heard everybody saying "Yes, yes" to positive over negative. But not "Yes, yes" to positive noninvasive always over an invasive mode of positive pressure ventilation in all situations.

Bach: Well, the initial attempt should be with noninvasive systems. I think we all said yes to that.

Hardy: To an acute respiratory failure?

Pierson: The statement I was making was that when noninvasive mechanical ventilatory support is to be used, it should be positive pressure.

Kacmarek: We've already made the commitment to noninvasive.

Hardy: OK. Great! Point 2 was in answer to Barry's (Make) question, which was, Are there situations where we're likely to extubate somebody and expect problems? Of course. We could all think of a number of situations where that's true, and I think the reason there wasn't a lecture on that topic is that this was supposed to be controversies in home management, and we're spending a lot of time actually talking about acute failure, hospital management, ventilatory situations, that kind of stuff.

Hardy: There are certain situations like facial trauma and burns to the face where you're going to have difficulty with any facial mask.

Make: That's your clinical expectation, but are there any data on that point?

Hardy: True. There is no study I know of specifically addressing extubation to noninvasive mask for children.

Kacmarek: I want to comment on Barry's (Make) statement about face mask ventilation. I disagree, I think there are a lot of reasonably good face masks available now. Not the black anesthesia masks but a number of clear masks are available that can be used. I think, particularly in the hands of somebody who is inexperienced in noninvasive ventilation, it's easier to use an ICU ventilator with a face mask than it is to take a pressure-limited machine and try to use a nasal mask because of all the issues of sizing and air leak particularly in the acute setting.

Make: I think that'll be true in a year or two. I'm not so sure that everyone is comfortable with that therapy at the moment.

Kacmarek: Puritan-Bennett has a very nice face mask. There are 5 or 6 other companies that have really reasonable face masks that can be used. In the acute setting, there are more data to support the use of full face masks than there is supporting nasal masks.[1,2]

REFERENCES

1. Brochard L, Isabey D, Piquet J, Amoro P, Mancebo J, Messadi AA, et al. Reversal of acute exacerbations of chronic obstructive lung disease by inspiratory assistance with a face mask. N Engl J Med 1990;323(22):1523-1530.
2. Meduri GU, Conoscenti CC, Menashe P, Nair S. Noninvasive face mask ventilation in patients with acute respiratory failure. Chest 1989;95(4):865-870.

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Home Mechanical Ventilatory Assistance for Infants

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Introduction

It appears that the number of infants receiving mechanical ventilatory support at home is increasing, and, in my opinion, this increase parallels the increases seen in the number of adults receiving ventilatory assistance at home. Improvement in the overall management of the compromised newborn and advancements in respiratory support and monitoring have contributed to decreased infant mortality and to the numbers of infants transferred home, but continually intensifying cost-containment efforts have resulted in much sicker infants being discharged home with ventilatory assistance than occurred just 5 years ago. In addition to cost containment, the improved quality of life, enhanced development for the infant cared for in the home, and greater normalization of family life have also contributed to the increased numbers of infants maintained on ventilatory support in the home.[1-3] Technical problems associated with specific types of ventilator and approaches to ventilatory support are common. Controversy also exists regarding the level of monitoring required in the home and the qualification of caregivers assisting these infants. In this paper, I review these and other issues associated with home ventilatory assistance of infants in light of the literature and my own experience.

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Candidates for Home Ventilation

Infants with one of a number of diseases giving rise to respiratory limitation may be considered candidates for home ventilatory assistance. Most candidates can be classified as having chronic respiratory failure as a result of (1) ventilatory muscle weakness or skeletal deformity, (2) inappropriate neurologic control of ventilation, or (3) a primary cardiopulmonary disorder (Table 1).[4] In my opinion, infants with primary cardiopulmonary disorders are the most challenging of these groups. These infants frequently require high fractional inspired oxygen concentrations (F_{IO_2}s, 0.35 to 0.45) and positive end-expiratory pressure (PEEP) to improve oxygenation or stabilize diseased airways, and frequently are weaned from ventilatory support while at home.[3,5] Except in children with cardiac defects, the primary disorder is usually age-limited, improving over time because of continued growth and development of the respiratory system.

Table 1. Diagnoses of Infants Considered Candidates for Home Ventilatory Assistance

- Ventilatory muscle weakness
  - Muscular dystrophies
  - Congenital childhood hypotonias
  - Spinal cord atrophy
  - Myasthenia gravis
  - Phrenic nerve paralysis
- Thoracic deformities
  - Kyphoscoliosis
- Disorders of central control of breathing
  - Central hyperventilation syndromes
  - Ondine's curse
  - Arnold-Chiari malformation
- Primary cardiopulmonary disorders
  - Bronchopulmonary dysplasia
  - Pulmonary hypoplasia
  - Bronchopneumonia
  - Congenital heart disease

Infants with diseases affecting neurologic control of ventilation are normally the least technically complex to manage at home; however, these infants can be expected to require ventilatory support for life.[6,7] Because they have no primary lung disorder, neither PEEP nor high F_{IO_2} are required. In fact, many do not require oxygen supplementation and because ventilatory support is expected to be necessary for life, weaning is not a consideration.

Infants who have diseases that directly affect the function of ventilatory muscles or who have skeletal deformities occupy the middle ground between the first two groups. Their ventilatory needs may be minimal because of a lack of intrinsic lung disease, although with age (particularly in infants with kyphoscoliosis or muscular dystrophies) gas exchange may become a greater problem.[6,8,9] However, some infants in this category are capable of being weaned from ventilatory support as they develop.[8]

Clinical Discharge Criteria

Medical criteria for discharging an infant who requires ventilatory assistance to the home have become less confining over the years. With shorter and shorter hospital stays having become the rule, acceptable criteria for discharge have become more variable. In our institution in the past, infants less than 6 months of age requiring ventilatory assistance were never considered for discharge to the home, whereas recently we have discharged infants with "Ondine's Curse" who were as young as 3 months old with continual (24 hour/day) ventilatory support.

Table 2 lists global criteria for the clinical stability necessary for consideration for discharge to the home.[4] Of these, stable cardiopulmonary function and progressive growth and development are generally considered to be the most important and absolute criteria. Specific requirements regarding level of ventilatory support are flexible. Although it is ideal that the infant not require high F_{IO_2} or PEEP, some infants are discharged home with high oxygen requirements and PEEP. However, as a general rule,

Table 2. Criteria that Indicate Clinical Stability and Readiness for Discharge to the Home with Ventilatory Assistance*

- Overall medical stability
- Cardiovascular stability
- Ability to protect airway and clear secretions
- Evidence of progressive growth and development
- Frequent ventilator adjustments unnecessary
- Stable blood gases with $F_{IO_2} < 0.40$?† and PEEP = 0?

*Adapted from Reference 10.

†Some infants with high F_{IO_2} and PEEP requirements are discharged.

ventilator settings should be stable and the $F_{IO_2} < 0.40$. Frequent adjustment of the ventilator should not be necessary. Because periodic alterations in bronchial tone in these infants can result in instability, bronchospasm should be under control. Other system problems should be controlled and relatively stable.

A distinction needs to be made between the infant with a terminal disease whose family prefers that the infant spend the last weeks of life with the family and the viable infant whose prognosis is good. Criteria for discharge of the terminally ill infant with no life expectancy may differ greatly from discharge criteria for the infant who is expected to survive into adolescence and beyond.

Discharge Planning

In addition to medical readiness for discharge to the home, issues to be considered include the family's ability to cope with an infant requiring ventilatory assistance and the financial stresses placed on the family in this setting. Finally, the requirements for the infant's caregivers must be carefully considered.

The Family

Table 3 lists the characteristics of the family ideally suited for caring for a ventilator-assisted infant in the home. This table was originally developed by Gilmartin[10] to define the characteristics of adult ventilator-dependent patients being considered for discharge, but as it has been modified it also defines the ideal characteristics of the family in this setting. The placement of a chronically ill child in the home, regardless of the disease, markedly stresses all aspects of family life.[11] These stresses are compounded when the child is maintained on life support equipment. Families with pre-existing social or financial problems are not likely to survive intact after the placement of a ventilator-assisted infant into the home.[12,13] However, even with the ideal family, the disruption and change in family dynamics caused by a ventilator-dependent infant can be disastrous. During discussions about discharge, consideration should be given to siblings. We all know that it is difficult enough for siblings to accept a healthy new addition to the family because of the shifting focus of attention. The addition of a child

Table 3. Characteristics of Family and Parents of Infants Discharged Home on Mechanical Ventilation That Determine Success or Failure

Characteristic	Ideal	Acceptable	Unacceptable
Parental coping	Optimistic Motivated Resourceful Flexible Adaptable Sense of humor Directive	Optimistic Motivated Has a sense of humor	None
Support systems	Close family and social supports	Social supports adequate	Lack of family and supports
Parental education	College degree Able to learn	Able to learn Mechanically astute	Altered mental status Unable to learn
Financial resources	Adequate family assets Optimal health insurance	Adequate health insurance coverage	Lack of personal assets No health insurance
Infant medical condition	Stable neuromuscular disease, ventilator-free time No other medical illnesses	Stable neuromuscular or obstructive airway disease Limited or no ventilator-free time	Medically unstable
Infant-care ability of parents	Able to provide infant care and/or direct others	Able to provide infant care	Unable to care for infant or direct others

who requires the care that a ventilator-assisted infant requires can markedly affect the other childrens' sense of self-worth and cause them to act out and be disruptive. Time must be set aside to discuss the new sibling and the demands that the infant places on the family, but it is equally important to reinforce the love that parents have for all siblings. The siblings of a ventilator-assisted child must also feel that they are special.

Family Financial Capabilities

Depending upon the actual medical needs of the child, the cost of maintaining an infant at home can equal or exceed the cost of maintaining that child in a non-ICU, acute care bed.[14,15] Table 4 lists the primary factors that contribute to this high cost. It is imperative that careful analysis of insurance coverage and the financial capabilities of the family be conducted prior to the development of detailed plans for discharge.

Table 4. Factors Adding to Monthly Cost of Home Ventilatory Assistance for Infants

Nursing personnel
Non-nursing caregivers
Mechanical ventilator equipment
Oxygen
Other equipment (eg, suctioning)
Medication
General nursing supplies

Caregivers

Most of the cost associated with maintaining a ventilator-assisted infant in the home is related to the payment of caregivers.[14] Because few of these infants have any ventilator-free time at the time of discharge, I believe that it is imperative that 24-hour, 7-day/week caregiver support from outside the family be provided initially. This is not to exclude the family from caregiver activities but to allow for an appropriate transition from total to partial dependence on outside caregivers, based on the medical demands of the infant and the capabilities of the family. In my experience, it is impossible to predict how long 24-hour/day outside caregiver support will be necessary. In general, at least 6-12 months of 24-hour support is required for ventilator-dependent infants. Beyond this time, the individual capabilities of the patient and family determine need. In most cases 12- to 16-hour support is required until the child is at least school age (5-6 years old).

The qualifications of the caregivers who provide the 24-hour support is open to considerable debate. It is difficult to argue against care provided by registered nurses (RNs) on a part-time basis varying from daily to weekly interactions, depending on the demands of the child. But, it is also difficult from the perspective of medical necessity or cost to argue for 24 hour/day coverage by RNs. Most infants require little more than the standard care provided any infant plus airway suctioning, monitoring of equipment operation, and basic patient assessment. These activities can be taught to home care assistants and provided at a small fraction of the cost of an RN. In some States, Visiting Nurse Associations provide RN coverage at a cost of about \$30-\$50/hour, averaging about \$7,000/week for 24 hour/day coverage, whereas home care assistants frequently can be hired for about \$8-\$12/hour (costing about \$1,750/week). Even if RNs provide care for a few hours daily, the cost of caregiver assistance can be reduced by more than 50% if trained assistants are used. Of major concern is the fact that many third-party payers provide reimbursement only for RNs and will not provide reimbursement for unlicensed caregivers. This position has kept the cost of home ventilatory assistance for infants unnecessarily high and, in many cases, prohibitive. I believe that it is imperative for the continued success of infant home ventilatory assistance programs that third-party payers reconsider their position on reimbursement of nonprofessional or unlicensed caregivers.

Transition to the Home

The transition of any ventilator-assisted person into the home is complex, but when the person is an infant the complexity increases because of the inability of the infant to participate in the process and the lack of equipment designed specifically for infants. The process should begin as soon as it becomes clear that the infant may be a candidate for home ventilatory assistance.[14] Generally, at least 4 weeks is needed between the time the decision is made and the actual transfer occurs. If the child is not medically stable at the time the decision is made, this minimal time period may be longer. During this period, financial problems must be addressed and solved. No child should be transferred before all financial issues are settled. All equipment that is to be used in the home should be used by the infant while in the hospital.[5] That is, the specific ventilator with circuit that will be used at home should be used in the hospital. This is to ensure that all problems regarding the use of specific pieces of equipment are solved, and that family and all caregivers are familiar with their operation.

During the predischarge period, decisions regarding caregivers in the home should be made (that is, the percentage of the time that RN coverage is required and the percentage of the time coverage is to be provided by assistants). The specific individuals filling these roles should be selected, and all training ideally should be accomplished in the hospital before discharge. In some states, unlicensed caregivers may have to be trained by the family after discharge. As a result, family members must become experts in the care of their child before discharge and be comfortable with every aspect of the operation of ventilatory support equipment and of the performance of patient care procedures.

The whole process of making the transition to the home requires the coordination and cooperation of a team of healthcare providers as listed in Table 5 and discussed by Gilmartin in this issue.[16] It is ideal if over the month or two that the transition is occurring, a family member emerges as the coordinator of all activities regarding the care of the infant. In my experience, the most successful transitions are those in which a family member has assumed responsibility for direction of care.

Table 5. Primary Members of Discharge Planning Team

Patient's family
Attending physician
Social worker/continuing care coordinator
Primary nurse
VNA* nurse coordinator
Pediatric respiratory therapist
Home care therapist
DME supplier

*VNA = Visiting Nurse Association; DME = durable medical equipment.

Just prior to discharge, the home environment situation should be simulated. That is, all aspects of the child's care should be performed by those who will be caring for the child at home. This simulation normally identifies any last minute problems and builds the confidence of the family and caregivers.

Mechanical Ventilator

Unfortunately, no home mechanical ventilators are designed specifically for use by infants. All available models are designed for adults, although most may be used with infants. The decision that must be made is whether the discharged infant can be effectively managed with a home care ventilator or if a neonatal ICU ventilator is to be used. It has been my experience that any stable child can be managed with an adult home care ventilator but these machines have limitations. On the other hand, it is possible to set up a neonatal ICU ventilator in any home, but this greatly increases the complexity of home ventilatory assistance, increases cost, and markedly limits mobility[5] (Table 6).

The major technical differences between home care ventilators and neonatal ICU ventilators are in the method of gas delivery and in their ability to deliver oxygen and maintain PEEP.[15] The conventional neonatal ICU ventilator maintains a continuous gas flow and provides pressure-limited ventilation. Some newer neonatal ventilators do not provide continuous flow but rather provide flow-triggering that greatly improves their ability to provide assisted ventilation. The typical adult home care ventilator designed for invasive ventilatory support does not provide continuous flow, is relatively insensitive to patient inspiratory effort during spontaneous breathing, and imposes a high work of breathing.[17,18] Neonatal ventilators require oxygen, compressed air, and electrical power to function. Thus, either H-cylinders must be kept in the infant's room or the home must be piped for compressed gases. This greatly increases the cost and the complexity of home ventilatory support and normally limits the infant to one room of the house. Adult home care ventilators require only electrical power to operate, have internal batteries, and allow for reasonably accurate oxygen administration to levels of about 40-50%.[15] Considering the technical complexity, I rec-

Table 6. Comparison of Neonatal and Adult Home Care Mechanical Ventilators

Characteristic or Component	Neonatal ICU Ventilator	Adult Home Care Ventilator
Cost	\$14,000-\$18,000	\$7,000-\$9,000
Power to operate	Electrical & high pressure gases	Electrical
Portability	None	Portable
F_{DO_2}	0.21 to 1.0	0.21 to 0.40-0.50
PEEP	Built into ventilator	Must add on
Imposed WOB	Low	High
Internal battery	None	1-hour use
Alarms	Appropriate	Appropriate
Ease of operation	User-unfriendly	User-friendly
Use with increasing age	Limited as child grows	Usable infancy to adulthood

ommend an adult home care ventilator regardless of the situation. The only setting in which it seems even reasonable to consider a neonatal ICU ventilator, is when the anticipated length of home mechanical ventilation is limited (eg, 1-2 months) because it is expected that the child will be weaned rapidly or life expectancy is limited. However, it must be remembered that the cost of the ventilator system may more than double when neonatal ICU ventilators are used.

The question of whether bi-level positive airway pressure devices can be used arises during discussions of neonatal ventilation. The answer to this question today is No because none of the models available in the United States is designed for invasive use. None incorporates alarms, internal batteries, or mechanisms to accurately monitor volumes or pressures. However, the inherent design of these units is ideal for neonatal ventilation. They provide continuous flow and pressure-limited ventilation, and impose little work during patient triggering.[19] It is unfortunate that no manufacturer has been willing to redesign these devices in a manner that would make them suitable for invasive use by infants in the home care setting.

Mode of Ventilation

Historically, neonatal ventilation has been accomplished in the intermittent mandatory ventilation (IMV) mode. If a neonatal ICU ventilator is to be used in the home, the use of IMV normally does not present a problem (other than excessive gas consumption). However, caution must be exercised if IMV via an adult home care ventilators is contemplated. As illustrated in Figure 1, the typical home care ventilator is operated by a piston without incorporation of a demand system or continuous gas flow.[17] The suggestion that the synchronized IMV, or SIMV, mode is available on these ventilators is an incorrect representation of the operation of the ventilator. During SIMV with the adult home care volume ventilators available in the United States, the mandatory breath is an assist/control breath as with adult ICU ventilators. However, there the similarity between these devices ends. During spontaneous breathing periods, the infant must actively draw gas through the exhalation valve, the piston chamber, or the internal anti-suffocation valve (not available on all models). As a result, the imposed work of breathing with these units may be excessive

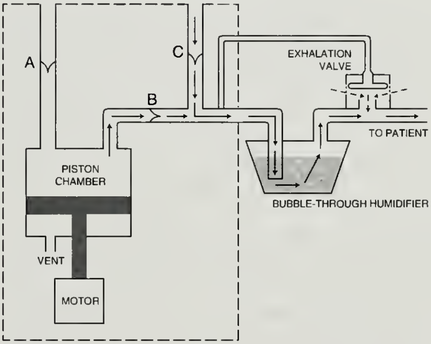

Fig. 1. Diagram showing gas flow through a typical home care ventilator. A. One-way check valve to allow gas entry into the piston chamber during the piston backstroke. B. One-way check valve to prevent subatmospheric pressure from developing in the circuit during piston backstroke. C. One-way check valve (anti-suffocation valve) to allow patient to inspire spontaneously during closure of B when piston backstroke is in progress. Some gas may be inspired through the exhalation valve during spontaneous breathing. (From Reference 17, with permission.)

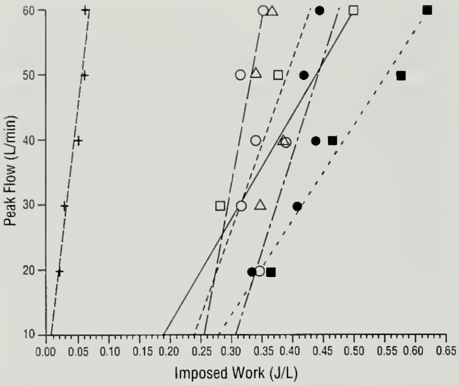

Fig. 2. Imposed work of breathing vs inspiratory flow for five home care ventilators with a normally functioning Cascade I humidifier (bubble-through) and an H-valve continuous-flow system modified with a Cascade humidifier (tower removed) used as a passover humidifier. All points represent mean values, and lines are linear regressions. Continuous-flow system with passover humidifier = + and —-— ; Bear 33 = ○ and —— ; Puritan-Bennett 2800 = △ and ----- ; Aequitron LP-6 = □ and ——; Medimax ARF-1500E = ● and —-—; and Lifecare PLV-100 = ■ and - - - - -. (From Reference 17, with permission.)

(Fig. 2).[17] Because of this, the SIMV mode can never be recommended with these ventilators without modification. If IMV is required, a secondary continuous-gas-flow system should be added to ensure that work of breathing is not increased by the ventilator (Fig. 3). Although this alteration provides appropriate ventilatory support, it markedly increases the complexity of the setup, limits portability, and increases the cost of home ventilatory support because of the high consumption of compressed gas.

When adult home care ventilators are used to ventilate infants, we customarily use the assist/control mode, focusing more on periods of ventilator independence than on providing partial ventilatory support, by decreasing the IMV rate. We ensure that pressure-limited ventilation is provided either by using an adult home care ventilator that allows pressure-limited plateau ventilation (Puritan-Bennett 2801 or Aequitron LP-10) or by adding a pressure-relief valve to the inspiratory limb of those ventilators that do not (PLV-100, PLV-102, and Bear 33). With either of these approaches, pressure-control ventilation can be simulated. The maximum plateau pressure can be set, as can the inspiratory time (either directly or by setting tidal volume, or V_T, and peak flow). As with all forms of pressure-control ventilation, V_T can be expected to vary somewhat on a breath-to-breath basis. In general, V_T delivery is set high so that some volume is always released by the pressure-relief (pop off) valve. This ensures that in periods during which a large airleak exists around the tracheotomy tube because of the patient's position, an adequate V_T is still delivered but also ensures that during periods of no airleak around the cuff excess volume is leaked from the circuit by the pop-off valve. When this approach is used, the high-pressure alarm should be set no more than 5

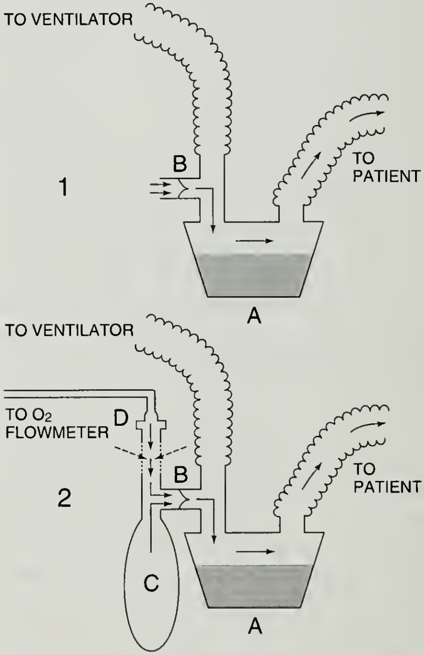

Fig. 3. Diagram of one-way H-valve systems: (1) Valve open to atmosphere; (2) valve with 3-L reservoir bag attached to 28% oxygen air-entrainment valve. Arrows indicate direction of gas flow during spontaneous inspiration. Setup 1 is used if no supplemental oxygen required. Setup 2 allows for the filtration of oxygen into the system. (From Reference 17, with permission.)

cm H_2O above the pressure plateau level so that transient spikes in pressure occurring with coughing or airway obstruction trigger the high-pressure alarm. Unfortunately, no home care ventilator and few neonatal ventilators monitor delivered V_T; thus, careful observation of chest movement to ensure volume delivery is required.

Control of Oxygen Concentration

With all home care ventilators except the PLV-102, precise oxygen concentration delivery (F_{DO_2}) is problematic.[15] The PLV-102 incorporates a proportional valve that allows fairly accurate F_{DO_2} up to 0.90. Other home care ventilators depend upon oxygen reservoirs attached to the air intake port to provide supplemental oxygen. With these attachments, accurate F_{DO_2} up to about 0.40 to 0.50 is possible, provided no change in respiratory rate occurs. However, as the rate increases F_{DO_2} can be expected to decrease. We have not, however, experienced major problems maintaining adequate F_{DO_2}s with home care ventilators during assistance to neonates.

PEEP

Fortunately the use of PEEP during mechanical ventilation of infants in the home is rarely indicated because usually only those infants with primary lung disease or unstable airways require it. If neonatal ICU ventilators are used, providing PEEP is not a problem. However, because PEEP is not an integral part of home care ventilators designed for invasive use, a PEEP valve must be added to the circuit. A number of problems arise if these valves are used. First, careful adjustment of the sensitivity control of the ventilator must be made. None of these ventilators is PEEP compensated, the sensitivity must be set about 0.5 to 1.0 cm H_2O below the PEEP level. That is, if 5 cm H_2O of PEEP is added, the sensitivity should be set at plus 4.5 cm H_2O to ensure patient cycling. If SIMV as designed on the ventilator is used, these PEEP valves markedly increase work of breathing because the PEEP level has to be overcome before gas can be inhaled from the piston chamber or exhalation valve. The same situation arises if a continuous gas flow is bled into the ventilator circuit (Fig. 3). PEEP must be overcome before the continuous flow can be diverted into the inspiratory limb. Finally, all add-on PEEP valves currently available function as variable flow resistors. As a result, they impose a high level of work of breathing to a spontaneously breathing patient's ventilator system.[20]

CPAP

A small subgroup of patients primarily with congenital airway abnormalities require long-term continuous positive airway pressure (CPAP) to stabilize their airways and maintain gas exchange. Increasing interest exists in sending these patients home. Of concern is the system used to establish CPAP. Because none of the commercially available nasal CPAP systems are designed for invasive use, such systems should not be applied to an infant with a tracheostomy tube. What is required is a continuous-flow CPAP system established by gas flow from compressed gas cylinders, air compressors, or gases piped into the home. These setups are technically demanding, expensive, and limiting. They require high gas flow, the use of a large-volume nebulizer or humidifier, and a loss-of-PEEP alarm. Of significant concern is potential disconnection of the large-bore tubing from the humidity source. If this occurs, not only is system PEEP lost, but rebreathing of exhaled CO_2 occurs. The use of a loss-of-PEEP (low-pressure) alarm is essential for the safe use of CPAP in the home.

Humidification

Adequate humidification must be provided to the infant, as with any patient requiring ventilatory support, to avoid retained secretions. Much discussion has occurred over the type of humidification system that is ideal for home ventilatory support; however, only a few conclusions can be drawn from the available literature. First artificial-nose humidifiers (heat-and-moisture exchangers, or HMEs) should not be used continuously on infants in the home care setting. Their humidification capabilities may be only marginal.[21,22] They can obstruct with secretions,[23] and their resistance to gas flow may be excessive.[24] In addition, because of the dead space inherent in the design, these devices are not recommended when tidal volumes are small (< 0.15 L) or when large leaks exist around the artificial airway.[25,26] As a result, I recommend these devices only for short-term periodic use in bigger children when away from home. I have found the other humidifiers in common use to function relatively equivalently.[27] Cost and convenience become more of an issue than performance, but cost is always greater with models that require the purchase of replacement wicks or specially packaged water. Although heated circuits avoid condensate in the circuit, they are more expensive than nonheated circuits and to my knowledge have not yet been shown to alter the risk of nosocomial infection. As a result, I recommend the use of a simple heated humidifier that can be filled with distilled water purchased in the local grocery store.

Artificial Airways

Frequent reassessment of the artificial airway of an infant requiring ventilatory assistance is essential to ensure continued adequate ventilation. Two primary factors differentiate infant airway care from that of adults. First tracheotomy tubes are not cuffed. As a result, the constant but variable leak must be taken into consideration when ventilatory support is adjusted. Second, because children normally grow rapidly during infancy, frequent size adjustment of the artificial airway is necessary.

In my experience, most infants require reassessment of the status of their airway and adequacy of ventilation every 2 to 3 months. This can usually be accomplished by the use of pulse oximetry, end-tidal CO_2 monitoring, and the assessment of inspired and expired volume during periodic office or clinic visits. Although I do not recommend the use of these devices to document the adequacy of gas exchange in critically ill, unstable infants, they do provide reasonably accurate estimates of change in gas exchange in the stable ventilator-assisted child. Careful assessment and documentation of this data at the time of discharge provide a useful baseline to evaluate ventilator settings and adequacy of artificial airway size during follow-up visits. The frequency with which artificial airway size must be increased is difficult to specify because it is dependent on the actual age of the child at discharge and subsequent growth and development in the home. In general, the younger the child at the time of discharge to the home, the sooner and more frequent the need for artificial-airway size adjustment.

Monitoring in the Home

Another point of ongoing debate is the level of monitoring required of infants receiving ventilatory support or, for that matter, oxygen therapy in the home. Although safe and effective ventilatory support must not be compromised by an inadequate level of monitoring, the goal of home mechanical ventilatory support should not be to make the home a mini-ICU. All gas delivery systems attached to artificial airways that increase airway pressure should be monitored for both high and low pressure. This is to ensure that excessive pressure is not maintained in the airway and that increased impedance to ventilation, excessive system leak, and inability to establish minimal system pressure are rapidly recognized. In addition, the ventilator's monitor should detect periods of apnea and continuously evaluate ventilator overall function. Whether continuous monitoring of F_{DO_2} is necessary depends on the system used, the F_{IO_2} required, and the probability of inadvertent alteration or loss of supplemental oxygen delivery. As a general rule, periodic assessment of F_{DO_2} by the home care therapist is more than adequate, provided the caregivers in the home are properly instructed in the setup and maintenance of oxygen delivery equipment.

As a general rule, the intent is to avoid creating an ICU in the home. Thus, decisions on adding monitoring equipment beyond those incorporated in the ventilator should be made cautiously. As in the ICU, the more monitors, the more false-positive alarms and the greater the conditioning of caregivers to delay response or to ignore any alarm.[28]

Apnea monitors have a role in nonventilator-dependent patients but perform a redundant function in ventilator-dependent patients. As discussed earlier, only home care ventilators that monitor high and low airway pressure and apnea should be used. Thus, in these patients the apnea monitor provides a secondary, unnecessary, and costly addition to an appropriately monitored system.

The greatest controversy regarding monitoring of patients on home ventilatory assistance may be whether continuous pulse oximetry is necessary. Before discussing the clinical issues, it is important to emphasize that pulse oximeters are inaccurate. For most units, the 95% confidence interval is ±4 to 6%.[29-31] That is, when a pulse oximeter reads 92% saturation, the patients actual saturation could be as low as 86% or as high as 98%. In addition, motion artifact can lead to frequent false-positive alarms. An additional concern is what is the home caregiver to do when the reading is above or below the targeted value? Should caregivers be provided with detailed algorithms outlining when, how much, and for how long F_{DO_2} should be adjusted if limits are exceeded? Or should the physician or home healthcare provider be called every time the limit is exceeded? This level of detailed micromanagement based on an imprecise indicator of patient status produces anxiety in caregivers and lacks documented benefit for the patient. To my knowledge, even in the ICU the use of continuous oximetry has not been shown to minimize adverse events and decrease morbidity.

I do not recommend the use of pulse oximetry on any ventilator-dependent patient who does not have a high F_{IO_2} requirement. Even if the F_{IO_2} requirement is high, the potential benefit of continuous pulse oximetry must be weighed against cost, complexity, and the anxiety it may produce. In those patients with high F_{IO_2} requirements, specific treatment protocols ensuring F_{IO_2} adjustment during stressful activities can be part of the patient's routine care plan, thus eliminating the need for continuous monitoring. Addressing situations that may precipitate adverse patient response eliminates the need for this inaccurate, costly, and potentially anxiety-producing monitor.

A comment regarding end-tidal CO_2 ($etCO_2$) monitoring in the home is necessary for completeness. Because I find it difficult to justify the need for continuous $etCO_2$ monitoring of any hospitalized ventilator-dependent patient, it is very difficult for me to conceive of a situation in the home where continuous $etCO_2$ monitoring is indicated. Arguments similar to those regarding pulse oximetry can be made regarding $etCO_2$ monitoring.[32-34] In addition, when the status of the airway and the ventilator settings are frequently re-evaluated, to my knowledge, no data suggest that monitoring of $etCO_2$ enhances the quality or safety of ventilatory assistance in the home.

Equipment Requirement

The equipment needs of ventilator-assisted infants are generally the same as those of ventilator-assisted adults: mechanical ventilator, manual ventilator, oxygen delivery system, humidification system, aerosol drug delivery system, and suctioning equipment.[14] In addition, it is imperative that a backup ventilator of the same type as the principal ventilator be present in the home. Because almost all infants sent home with ventilatory support require 24 hour/day support, the absence of a backup ventilator in the event of a ventilator failure is potentially disastrous.

Weaning from Ventilatory Support

In general, weaning from long-term ventilatory support is only an issue in those infants ventilated for primary respiratory disease. Most patients with central drive or neuromuscular/neurologic deficiencies cannot be weaned from ventilatory support, although they may make a transition to other approaches to ventilatory support as they grow and develop. In the home care setting, I always recommend T-piece trials as the approach to weaning. Because weaning in these infants may take months or years, full ventilatory support, interspaced with periods of increasing ventilator independence, should be the routine. In addition it should be remembered that SIMV administered through home care ventilators is plagued with problems, with the primary concern being imposed work of breathing.

If weaning is to take place in the home, evaluation of the weaning schedule should occur in the clinic or outpatient area before implementation in the home. This generally is essential to ensure patient tolerance of the protocol. In addition, careful instruction must be given to caregivers, to ensure rapid recognition of intolerance of the protocol. The frequency of clinic or office visits during this period generally depends on the infant's tolerance of the weaning protocol and the anticipated speed with which complete ventilator independence will be attained.

REFERENCES

1. Schreiner MS, Donar ME, Kettrick RG. Pediatric home mechanical ventilation. Pediatr Clin North Am 1987; 34(1):47-60.
2. Keens TG, Jansen MT, DeWitt PK, Davidson-Ward SL. Home care for children with chronic respiratory failure. Semin Respir Med 1990;11:269-281.
3. Keens TG, Jansen MT, DeWitt PK, Davidson-Ward SL, Bowman CM, Lew CD, Platzker ACG. Outcome of children treated for chronic respiratory failure by mechanical assisted ventilation at home. Am Rev Respir Dis 1991; 143(4, Part 2):A504.
4. O'Donohue WJ Jr, Giovannoni RM, Goldberg AI, Keens TG, Make BJ, Plummer AL, Prentice WS. Long-term mechanical ventilation: guidelines for manage- ment in the home and at alternate community sites. Report of the Ad Hoc Committee, Respiratory Care Section, American College of Chest Physicians. Chest 1986;90(1, Suppl): 1S-37S.
5. Kacmarek RM, Thompson JE. Respiratory care of the ventilator-assisted infant in the home. Respir Care 1986; 31(7):605-614.
6. Oren J, Kelly DH, Shannon DC. Long-term follow-up of children with congenital central hypoventilation syndrome. Pediatrics 1987;80(3):375-380.
7. Weese-Mayer DE, Silvestri JM, Menzies LJ, Morrow-Kenny AS, Hunt CE, Hauptman SA. Congenital central hypoventilation syndrome: diagnosis, management, and long-term outcome in thirty-two children. J Pediatr 1992;120(3):381-387.
8. Davidson-Ward SL, Keens TG. Ventilatory management at home. In: Ballard RA, ed. Pediatric care of the ICN graduate. Philadelphia: WB Saunders Co, 1988:166-176.
9. Gilgoff IS, Kahlstrom E, MacLaughlin E, Keens TG. Long-term ventilatory support in spinal muscular atrophy. J Pediatr 1989;115(6):904-909.
10. Gilmartin ME. Long-term mechanical ventilation: patient selection and discharge planning. Respir Care 1991; 36(3):205-216.
11. Aday LA, Wegener DH. Home care for ventilator-assisted children: implications for the children, their families, and health policy. Child Health Care 1988;17:112-120.
12. Murphy KW. Stress and coping in home care: a study of families. In: Hochstadt NJ, Yosh DM, eds. The medically complex child—the transfer to home care. New York: Harwood Academic Publishers, 1991.
13. Keens SE, Jansen MT, Lipsker LE, Keens TG. Marital discord and increased stress in parents of ventilator assisted children at home (abstract). Pediatr Res 1990;27: 11A.
14. Gilmartin ME. Long-term mechanical ventilation outside the hospital. In: Pierson DJ, Kacmarek RM, eds. Foundations in respiratory care. New York: Churchill Livingstone, 1992:1185-1204.
15. Kacmarek RM, Hess D. Equipment used for home ventilatory support. In: Tobin M, ed. Mechanical ventilation. New York: McGraw-Hill (in press).
16. Gilmartin ME. Transition from the intensive care unit to home: patient selection and discharge planning. Respir Care 1994;39(5):456-480.
17. Kacmarek RM, Stanek KS, McMahon KM, Wilson RS. Imposed work of breathing during synchronized intermittent mandatory ventilation provided by five home care ventilators. Respir Care 1990;35(5):405-414.
18. Hirsch C, Barker S, Kacmarek RM, Robart P. Inspiratory work of breathing imposed (WOBI) by the BiPAP™ ventilator (abstract). Respir Care 1992;37(11):1315.
19. Kacmarek RM, Robart P, Hirsch C, Barker S. Work of breathing imposed during spontaneous breathing in the SIMV mode of home care ventilators (abstract). Respir Care 1992;37(11):1358,1360.
20. Kacmarek RM, Mang H, Barker S, Cycyk-Chapman MC. The effects of disposable/interchangeable PEEP valves on work of breathing during CPAP. Crit Care Med (in press).
21. Branson RD, Hurst JM. Laboratory evaluation of moisture output of seven airway heat and moisture exchangers. Respir Care 1987;32(9):741-747.
22. Eckerbom B, Lindholm CE. Performance evaluation of six heat and moisture exchangers according to the Draft International Standard (ISO/DIS 9360). Acta Anaesthesiol Scand 1990;34(5):404-409.
23. Martin C, Perrin G, Gevaudan MJ, Saux P, Gouin F. Heat and moisture exchangers and vaporizing humidifiers in the intensive care unit. Chest 1990;97(1):144-149.
24. Ploysongsang Y, Branson R, Rashkin MC, Hurst JM. Pressure flow characteristics of commonly used heat-moisture exchangers. Am Rev Respir Dis 1988;138(3): 675-678.
25. Hess D, Kacmarek RM. Technical aspects of the patient-ventilator interphase. In: Tobin M, ed. Mechanical ventilation. New York: McGraw-Hill (in press).
26. Martin C, Perrin G, Gevaudan MJ, Saux P, Gouin F. Heat and moisture exchangers and vaporizing humidifiers in the intensive care unit. Chest 1990;97(1):144-149.
27. Kacmarek RM. Humidity and aerosol therapy. In: Pierson DJ, Kacmarek RM, eds. Foundations of respiratory care. New York: Churchill Livingstone, 1992:793-824.
28. Hess D. Noninvasive monitoring in respiratory care—present, past, and future: an overview. Respir Care 1990;35(6):482-499.
29. Kelleher JF. Pulse oximetry. J Clin Monit 1989;5(1):37-62.
30. Tremper KK, Barker SJ. Pulse oximetry. Anesthesiology 1989;70:98-108.
31. Ries AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. Am Rev Respir Dis 1985;132(3):685-689.
32. Withington DE, Ramsay JG, Saoud AT, Bilodeau J. Weaning from ventilation after cardiopulmonary bypass: evaluation of a noninvasive technique. Can J Anaesth 1991;38(1):15-19.

33. Hess D, Schlottag A, Levin B, Mathai J, Rexrode WO. An evalaution of the usefulness of end-tidal P_{CO_2} to aid weaning from mechanical ventilation following cardiac surgery. Respir Care 1991;36(8):837-843.

34. Smith RA, Novak RA, Venus B. End-tidal CO_2 monitoring utility during weaning from mechanical ventilation. Respir Care 1989;34(11):972-975.

Kacmarek Discussion

Celli: My question is, What's the outcome with these kids? I'm just an 'adult' doctor here, and I would like to hear from you what happens to these kids down the road after they go home?

Kacmarek: We have kids followed in our pediatric pulmonary clinic who are graduating from high school, who have been on home mechanical ventilation since birth because they don't breathe when they go to sleep. Now, they make the transition to different levels of ventilation over time. Some come to tolerate noninvasive ventilation (although a lot of them who have had the tracheostomy since birth are a little more difficult to transition away from the tracheostomy). To be quite specific, we've had only the rare patient willing even to consider noninvasive ventilation. Dan Shannon's patients have gotten to the point where they're graduating from high school, living a relatively normal life. Once they get a little bit older and you can reason and interact with them, you can plug their trachs—we actually take the tube out during the day and put a plug in that keeps the stoma open. The child spends the whole day in school with unassisted children—they interact and they talk. They simply have to remember that they can't take naps in school. They come home, and at night they go on mechanical ventilatory support. Otherwise they live a very normal life. That's one extreme; the other extreme is the child who requires short-term mechanical ventilation because of BPD (bronchopulmonary dysplasia) or other acute conditions, who eventually weans from ventilatory support and goes on to normal existence.

Pierson: Over what period of time and at about what age?

Kacmarek: With BPD it really depends. You'll find some children who wean at a couple of months, some at a year, 18 months, or even longer. Karen may be able to add to that.

Hardy: I enjoyed that presentation greatly—a good summary of something that pediatric pulmonologists do regularly. The populations include long-term ventilation for children with abnormal control of central alveolar hyperventilation, previously called Ondine's Curse. Caregivers, trained attendants, are important for these children. For children in kindergarten, naps are a serious problem. These kids get tired at school, and they will fall asleep and need support. They also can have little traumas that can make them apneic. They fall off the swing set, for example, and they do not have normal control of ventilation. A minor hypoxemic event can make them stop breathing—totally. Short-term ventilation needs are more common for premature infants who develop BPD. Neonatologists are continuing to resuscitate smaller and smaller newborns, and the likelihood of your having BPD if you're under 1,250 grams as a neonate is extremely high. I would not have you all believe that those babies are just universally being discharged. They usually stay in the intensive care nursery setting, as opposed to being a candidate to go home. BPD does occur in premature infants > 1,250 grams birthweight, who more quickly become candidates to go home, as you've just heard Bob (Kacmarek) describe.

Kacmarek: But even those children that you've discussed—the little teeny ones (and I would agree with you, they don't get out of the hospital as rapidly)—most of them do get to the point, if they still require ventilatory support, where at 3 or 4 or 5 or 6 months, if they have the appropriate home environment, they could be transferred.

Hardy: Some babies are in intensive care nurseries from the time they are born and are ventilated for more than a year in the hospital. You can find them in any state in the United States. Ultimately, if ventilator dependent they become stable enough to undergo this transition, but you need a good support system. There aren't pediatric pulmonary centers in the United States willing to take a BPD baby on whom they've spent millions of dollars and send that baby home, unless the family has a very good support system. I'm sure we're going to hear more about that in a minute. Babies with spina bifida and a number of different neuromuscular problems also can be sent home on systems like this and usually go through the transition of different kinds of ventilatory support. Another category that you didn't discuss is the child who needs chronic CPAP in the home. I sent home the first infant in California who required continuous CPAP, supported by the state health program. I see many babies who have congenital airway abnormalities, airway malacia that is very severe, that involves the larynx and the trachea and often bronchi. These are the kind of babies who usually have multiple arrests in the first few weeks of life because they're obstructing, and they need positive pressure support in order to be able to move air. Pneumatic splinting of infant airways is something that has not been widely applied yet in pediatrics. I have two babies under 18 months of age at home on nasal positive pressure ventilation.

Kacmarek: There is nothing in the literature on this.

Hardy: I have to write up these cases, obviously.

Kacmarek: This is the first I've ever heard of a child that young on a noninvasive approach to ventilatory ..., other than negative pressure.

Bach: In Paris, Dr Anne Barois has over 100 patients with Type 1 or 2 spinal muscular atrophy (SMA). She has treated a number of these patients with nasal ventilation and, at times, mouthpiece ventilation over the last 20 years.[1]

REFERENCE

1. Barois A, Estournet B, Duval-Beaupere G, Bataille J, Leclair-Richard D. Amyotrophie spinale infantile [Infantile spinal muscular atrophy]. Rev Neurol Paris 1989;145(4):299-304.

Kacmarek: How young?

Bach: She describes patients using nasal ventilation from 15 months of age.

Hardy: You can do it in the specific instance of a baby who has a neuromotor problem. You've got to remember, they can't be wigglers. Babies who can't wiggle around, and who have a breathing problem can be put onto a mask system, and they'll do fine. They're not able to wiggle and move it or take it off.

Kacmarek: Creating a CPAP system in the home is not an easy thing to do. Are you using adult CPAP, continuous-flow generators? What are you using to try to establish this in an 18-month-old child?

Hardy: We used basically a routine kind of system, like you would have used in the hospital. We had H tanks in the house and a classic CPAP system set up in the household. This baby had severe airway malacia, had tracheostomy, had positive support, and I'm talking very positive, this kid needed, initially, CPAPs of around 25 cm H_2O. Well, it's rare, but it's definitely been done and there are a number of case reports of small numbers of infants who have had home CPAP support like this,[1-3] with pressures anywhere from 25, 35 cm H_2O, a lot of pressure to start, who, over time, gradually stiffen up their cartilage, in a period of years. You can usually guarantee parents that before kindergarten, the child will be out of this predicament. But most of the time the real severe kids are ventilated this way, with CPAP support for around 3 years. First you get them off of the positive pressure, and then after a while, their airways are stiff enough that you can get them decannulated, and they do fine. Now they're in school and they're running around. They're fine.

REFERENCES

1. deLorimier AA, Harrison MR, Hardy K, Howell LJ, Adzick NS. Tracheobronchial obstructions in infants and children: experience with 45 cases. Ann Surg 1990;212(3):227-289.
2. Wiseman NE, Duncan PG, Cameron CB. Management of tracheobronchomalacia with continuous positive airway pressure. J Pediatr Surg 1985; 20(5):489-493.
3. Sotomayor LJ, Godinez RI, Borden S, Wilmott RW. Large-airway collapse due to acquired tracheobronchomalacia in infancy. Am J Dis Child 1986; 140(4):367-371.

Kacmarek: The type of system that you are describing, though, really is limiting. I mean these children are really stuck to one room, unless they have very strong parents who can haul H cylinders around.

Hardy: The main system was in his bedroom on the first floor. He had an incredibly long tubing and could go throughout the living room and out the glass doors to a bit of the yard.

Stelmach: What was the oxygen concentration?

Hardy: He was never on much, maybe he was on 28% or so in the beginning and then just air. Initially, he had a few pneumonias, but his problem was the need for a pneumatic splint.

Hill: What information is available on what home mechanical ventilation of infants and children does to families?

Kacmarek: It's quite difficult dealing with families of any patient who's ventilator-dependent. There are a lot of families who don't end up being families when there's a child this sick going home with mechanical ventilation. It depends on the way the family is bound together to begin with. Those who have good family structure and good relationships will hold up, but if there's anything, if there's any reason to break up, this will do it. Sending a kid this sick home with mechanical ventilation almost always will cause a divorce, if there are already underlying problems.

Hardy: I would just add two other comments. One was about your monitoring question. I totally agree with you on that. I think that when you're sending a child home for home ventilatory support, they need to be very stable. This shouldn't be a child who needs a lot of extra monitors, other than the ones that are on your ventilator system. I never use ICU ventilators. We always use portable home ventilators. Second, I agree that the caregiver needn't be an RN—but a carefully trained individual. The hours of coverage are hotly debated by physicians and insurers. We always strive for 24 when they first go home. We are usually required to taper to 16 hours and then 12.

Kacmarek: My comment would be at least 12 hours—often more—of funded support. As you well know, dealing with insurance companies is a real problem. Getting these kids home costs an arm and a leg.

Dunne: A word of caution, however, if 12 hours appears in a document, it may become the standard, right or wrong!

Kacmarek: OK. OK. All I'm saying is that I think it depends on the whole family. You take a child with an extended family—a grandmother and grandfather of reasonable age, parents in the home, other siblings. Some families can handle a child without 24-hour a day support, but 24 hours is highly recommended in the majority of circumstances.

Hardy: Physicians in every state are trying to get adequate coverage and support, and yes, maybe sometimes they're going to amass some of their hours from family members. Situations exist where family support is not available and other trained individuals are absolutely necessary for these kinds of kids to go home.

Nickerson studied outcomes of infants who are home with tracheostomies in California. Home deaths of infants with tracheostomies in California only occurred when night nursing coverage was not available.

Kacmarek: I agree with you that night coverage is essential.

McIntyre: Good family structure and relationships have been identified as necessary for families to cope with the emotional and physical stress surrounding ventilator-dependent patients in the home. Are there support groups for these families?

Kacmarek: Yes, every center that sends children home usually has a support group of some type designed to allow the families to interact. It really is necessary. It's a tough transition for the family—although I don't think it's as tough as having this child in the hospital for 2 or 3 years, which circumstance is most destructive depends on the specific family.

Bach: I've had 6 SMA Werdnig-Hoffman Type-1 patients, who were on ventilators from birth or early infancy who were then transferred to our ventilator unit. Two of them are still alive, and one has been ventilator-supported for 15 years. The problem is that these patients cannot move at all or even communicate. We've never been able to figure out if they're intelligent. It's a real problem ethically, as well as financially. Does anyone else have experience with the Type-1 SMA patient, ventilator-dependent from birth? Once the diagnosis is made, should these patients be continued?[1]

REFERENCE

1. Wang TG, Bach JR, Alba AS, Avilez C. Prolonged survival by ventilatory support for individuals with spinal muscular atrophy. Am J Phys Med Rehabil (in press).

Kacmarek: We have never had a patient with that diagnosis. I don't know what the answer is, John. I don't think this group is set up to deal with specific ethical issues, although I think it's an important consideration with any child that's mechanically ventilated. The children that we primarily deal with will become normal adults. Their only long-term problem is that at night they have to be attached to a mechanical ventilator. I think the only right thing to do is to support them through infancy—til they get to the point where they can take care of themselves, and most do.

Decker: I want to ask a different question. You talked about having home care by RNs and LPNs. Looking at cost-effectiveness, could this be something else that other health professions could do?

Kacmarek: No, I meant nonprofessionals. I think it's ideal for the family to hire nonprofessionals. The problem they get into, as we discussed yesterday, is reimbursement. Unfortunately, the wisdom of insurers is that you only be reimbursed for the highest qualified person in the home. They're not willing to pay for a noncertified individual. All most need is somebody to take care of them in the true sense of being a babysitter, with enough intelligence to be taught basic skills and care of the child who's mechanically ventilated. There's not a lot of nursing here that's needed on a 24-hour basis. You may need a nurse to come in every day for 4 hours the first 3 months of life, but not 24 hours a day.

Make: There are two sets of Clinical Practice Guidelines for patients receiving mechanical ventilation outside the ICU that are currently being developed. One by the AARC and the other by the ACCP. In the ACCP discussion, the issue of alarms and monitors was raised by Tom Keens who recommends additional alarms, monitors, and oximeters in the home.

Kacmarek: We aren't finished yet.

Make: Others do not routinely use additional alarms. There certainly does appear to be some differences in clinical practice. Nobody has the information to make concrete recommendations.

Kacmarek: Which is the reason I raised it as a controversy. I will place it in the article as a controversy. However, my personal recommendation would be not to use it.

Make: I don't think anybody's had sufficient experience to be able to recommend this. The last question I had was a technical one. Could you explain your comment about the lack of reliability and stability of higher F_{IO_2}s?

Kacmarek: None of those systems is designed to deliver specific F_{IO_2}s, except the PLV-102. With most, the F_{IO_2} is a result of whatever flow you put into a reservoir. The higher the F_{IO_2} needed, the more difficult it is to achieve. As you start to see a patient's minute ventilation vary, the swings in the F_{IO_2} become even greater at higher F_{IO_2}s.

Make: Unless you had an accumulator or reservoir on ventilator intake and if tidal volumes are low, which they usually are in children, then might it be less of a problem?

Kacmarek: Sure. There's no question, but it still varies. I mean, you end up adding a fixed flow of 2 L/min, assuming a minute ventilation of 3 L, now that changes . . .

Make: Well, that's assuming you use a fixed flow of 100% oxygen.

Kacmarek: But you have to at one point in time, by the design of the systems. If I have a reservoir, I've got to set my flowmeter someplace, and if the child's minute ventilation changes, . . .

Make: You could bleed oxygen into a reservoir using a Venturi and might not need 100% oxygen.

Campbell: I had a comment about monitoring. I'm a little nervous in the hospital using pressure-limited time-cycled ventilation with only pressure monitoring for alarms. I don't work with infants, but I would think with those small tubes, just a small amount of secretions in there would make big changes in resistance and would dramatically affect their ventilation. I'd be a little bit nervous about trying to minimize the alarms and using that technique for ventilation.

Kacmarek: I understand what you are saying, although with appropriate humidification we've had no trouble with occlusion of the airway. I mean children do get sick, and you do get secretion accumulation, but we've never had an obstructed airway. Yes, with newborns, you're dealing with a 3- or 3.5-mm tube, but once you get past 3 or 4 months of age the tube is a little larger. I have not had any trouble with airway occlusion provided I have a good humidifier, which is why I'm totally against using any type of heat-and-moisture exchanger. They have them for little kids, but I would not ever recommend using them on these children.

Campbell: They do have a place, I think, if they're going outside the home, or if they're going . . .

Kacmarek: Once the child gets a little bigger and they're a little mobile, yes. But when they're tiny, all you do is get into trouble with them.

Leger: I am surprised that you recommend not using monitoring. Not "transferring the ICU to the home," as you said. I have not had a lot of experience with these children, but in recommending to maintain these patients at home if they are stable, how is it possible to define the stability of patients with tracheomalacia or a very small trach tube, easily obstructed or easily removed? These tubes are very small, and it requires little movement to push the tube out of the trachea.

Kacmarek: The big issue is the amount of anxiety that you create when you try to duplicate what's necessary in the hospital. I question the level of monitoring we do in the hospital, period. In the neonatal intensive care unit, for example, everybody has pulse oximeters on but rarely do staff pay attention to them. Monitors are not utilized anywhere near the level that they're designed to be used. I'm not saying that it's not needed in some patients, but in the majority of patients you do not need any additional monitoring. If a child requires extensive monitoring, they are not stable enough to be sent home.

Celli: The ACCP had a marathon session from 2 to 6 PM on monitoring in the ICU. The only thing I came out of that with was that none of those monitors, not one of them, has been shown to produce any beneficial effect of any kind in the ICU. Swan-Ganz, pH monitor in the stomach, nutrition, they talked about the whole thing. I sat through the whole thing, and I'm going to go home and take all the monitors out.

Kacmarek: End-tidal CO_2 monitors and oximetry are some of the worst, when you look at accuracy, reliability, and reflection of actual arterial values.

Masferrer: You probably have read the proceedings from a conference like this that we had a few years ago where we said the same thing.[1] Bob, besides the pressure plateau, what else should your ideal ventilator have?

REFERENCE

1. American Association for Respiratory Care. Consensus conference on the essentials of mechanical ventilators. Respir Care 1992;37(9):1000-1130.

Kacmarek: Ideally, it would be nice to have a home care ventilator for neonates. We have BiPAP™ units that can provide continuous-flow pressure-limited ventilation. All we need to do is to take the basic design of a compressor unit, modify it for an infant, add alarms and monitors, and we have an ideal infant ventilator for use in the home. I don't think it's real expensive. I think we have the basics for it in the bilevel CPAP-type ventilators. That's the kind of continuous-flow system we want that will allow us to set PEEP, that'll allow us to deliver plateau pressure ventilation.

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Collaborative Self-Management Strategies for Patients with Respiratory Disease

Barry Make MD

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Introduction

Healthcare professionals, including physicians, nurses, and respiratory care practitioners (RCPs), spend many years involved in their own education. Practitioners must complete undergraduate and often postgraduate formal training programs to improve their cognitive understanding of anatomy, physiology, and pathophysiology. They must then gain practical knowledge and experience in the technical skills necessary to care for patients. Because of the extensive knowledge of disease gained during their training, healthcare professionals are experts in understanding the appropriate therapy for the disorders presented by their patients. Society in general and patients in particular have generally shared this view—patients present with symptoms, and physicians order treatment. However, practitioners observed that patients did not precisely follow their treatment programs, and a large body of literature documented that patients were often not compliant with treatment. This model of healthcare and the term "compliance" not only recognize the expertise of the practitioner but also place the practitioner on a pedestal—on a level above the patient. The patient is seen as subservient to the higher authority of the practitioner, traditionally the physician, and is expected to comply with the physician's orders. This physician-patient relationship is similar to the relationship between a parent and a young child. However, alternative strategies that improve patient adherence and outcome are now available.

More recently, the relationship between healthcare professionals and their patients has matured and changed. Patients and practitioners, including physicians, are more frequently viewed as coequals in their relationship and as partners sharing the goal

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of reduced symptoms and improved health. The healthcare practitioner is viewed as a facilitator who assists in the patient's decision-making process. In this role, the facilitator must incorporate an educational component to assist the patient in making an informed decision.

Various terms may be used to describe the practitioner-patient relationship. Self-care connotes the use of preventive behaviors to enhance health by a healthy individual.[1, 2] Self-management may not be an optimal term because it implies that patients make decisions about their treatment in the absence of input from the practitioner.[3] The term collaborative self-management has been suggested because it embodies the concept of patient decision making with the assistance of the healthcare practitioner.[2] Co-management is also an appropriate term to describe collaborative self-management because it emphasizes the relationship between practitioner and patient as coequal in the management of the disease process. Unfortunately, these four terms (self-care, self-management, collaborative self-management, and co-management) are often used imprecisely and interchangeably and may denote only the use of limited forms of patient education.

The theoretical basis for co-management has been ascribed to Bandura's self-efficacy theory, which links the patient's knowledge and behavior.[4] Self-efficacy refers to the patient's expectations that a behavior will result in a positive outcome. For example, the strength of an asthmatic patient's belief that use of an inhaled bronchodilator will reduce dyspnea and alleviate symptoms determines whether that patient will use the medication. Education that enhances self-efficacy might be expected to improve collaborative self-management. Patients need to gain confidence that their symptoms can be reduced and that they can successfully co-manage their disease.

Underlying the concept of collaborative self-management is the principle of patient autonomy, which denotes the right of the patient to make his or her own decisions and choices regarding healthcare. For example, suppose a physician has prescribed an inhaled bronchodilator to be taken regularly four times daily by a patient with mild asthma. The patient may decide that he does not have sufficient symptoms to take the medication regularly or that the drug results in undesirable side effects such as hand tremors. Patient autonomy gives the patient the right to make the final decision regarding use of the medication. With the application of collaborative self-management, the patient would be encouraged to contact the physician, report the side effects, and discuss the potential choices for alternative management approaches such as lowering the dose of the medication, discontinuing the medication, or initiating a trial of a medication with potentially less side effects. Patient autonomy extends not only to daily decisions concerning such seemingly routine issues as taking prescribed outpatient medication for a chronic condition but also to life-and-death decisions regarding withholding and withdrawing mechanical ventilation and other life-supportive treatments. The American Thoracic Society (ATS) statement on withholding and withdrawing life-supportive therapy clearly recognizes the autonomy of the patient in making such choices even if those choices result in the eventual demise of the patient.[5] The ATS statement outlines other necessary components of the decision-making process to assist the patient in making an informed choice. A key component of self-management is education by the physician and other healthcare professionals in a manner that is readily comprehended by the patient. Informed consent requires that patients have the capacity to understand the choices presented and the consequences of their decisions. Once patients have reached an informed decision, physicians have the responsibility to act upon these decisions in a humane and compassionate manner.

Collaborative self-management is a relatively new concept that is gaining widespread acceptance by patients and the medical community but has been rigorously applied and investigated in only a few diseases. Because there is a relatively large body of published investigation on the co-management of asthma, I focus on self-management of this disease. Asthma presents unique challenges for self-management in the home and, because of the diverse management issues, can be considered a model for self-management of other chronic respiratory disorders. Because of the large number of studies of self-management in asthma, only a few of the published investigations that illustrate the most important findings are reviewed in detail.

Collaborative Self-Management in Asthma

Asthma is a relapsing and remitting disorder associated with airflow limitation that may lead to significant symptoms (eg, dyspnea, wheezing, cough). Patients with asthma may become worse while at home, requiring changes in management and medications. Uncontrolled disease may result in the need for acute care in the physician's office, in the emergency room, or in the hospital. There are multiple potential outcome measures of asthma management, including mortality, medical morbidity, respiratory symptoms, physiologic indicators of airflow limitation, quality of life, presence or absence of health-related behaviors, functional capacity for activities, utilization of healthcare resources, and other costs to the family and society, such as time lost from work (Table 1). Patients with asthma may also be relatively asymptomatic until the disorder is severe, thereby compounding the difficulty of self-management; asymptomatic patients may not recognize worsening disease and the need for changes in therapy to prevent the need for emergency care and hospitalization.

In 1991, the National Heart, Lung and Blood Institute (NHLBI) published "Guidelines for the Diagnosis and Management of Asthma."[6] Because the Guidelines were developed to represent the consensus of experts in the field and were widely distributed to healthcare practitioners in the United States, they have become the standard of care for these patients. The Guidelines clearly recognize the importance of collaborative self-management of asthma and the need for self-monitoring the degree of airflow limitation by routine patient measurement of peak flow in the home. The Guidelines note the importance of integrating education into continuing patient care for both adults and children.

Table 1. Potential Outcomes of Collaborative Self-Management in Patients with Asthma

- Mortality
- Morbidity
- Medical complications
- Respiratory Symptoms
  - Dyspnea
  - Wheezing
  - Cough
  - Symptoms during exercise
  - Symptoms at night
- Physiologic (airflow limitation)
  - Spirometry
  - Peak flow
- Quality of Life
  - Physical function
  - Mental & emotional function
  - Social function
  - Role function
  - Perception of well-being
- Health-Related Behaviors
  - Smoking
  - Sedentary lifestyle
- Functional Capacity
  - Able to work, attend school
  - Able to perform activities of daily living
- Utilization of Resources
  - Emergency room visits
  - Inpatient hospitalizations
  - Physician's office visits
- Other Costs
  - Direct costs of medical care
  - Indirect costs of medical care
  - Costs to family & others

Table 2. Components of Collaborative Self-Management

- Development of a patient-practitioner partnership that encourages communication and active patient participation
- Development of therapeutic goals mutually shared by patient and practitioner
- Instruction of the patient by the practitioner including written materials
  - Patient is ready and able to learn, process, and apply medical information
  - Information is presented so that it can be readily comprehended by the patient
  - Assessment of the patient's knowledge and skill
  - Discussion of the patient's concerns and fears
- Monitoring of collaborative self-management
- Family involvement when appropriate

The components of collaborative self-management are outlined in Table 2. However, studies reporting the results of co-management of asthma do not uniformly use all the components listed in Table 2. On the other hand, there are few data on the effects of employing the components of self-management singly. Almost all investigations include patient education, and the majority incorporate peak-flow monitoring. Although most studies report instructing and encouraging patients to act upon changes they detect in their asthma, only a few studies rigorously report specific instructions given to patients on how to effect these management changes. Because of the variability in the disease among patients, specific instructions must be tailored to the individual patient.

Physician Accessibility

As Table 2 indicates, an important component of collaborative self-management of asthma is the physician's willingness to commit to such care. Sufficient time should be allotted to patient appointments to reinforce the specific details of co-management for each individual patient. Physicians and other healthcare providers must be able to effectively communicate in an ongoing fashion with their patients to assure that collaborative self-management is effective. Teaching asthma collaborative self-management requires a basic knowledge of the signs, symptoms, and treatment of the disease and may be augmented by written and audiovisual materials. The NHLBI Guidelines for the Diagnosis and Management of Asthma[6] provide basic information on asthma management for healthcare professionals, and written educational materials for patients are available from the American Lung Association and some pharmaceutical companies. Information is also available to assist healthcare professionals in teaching asthma co-management.[7]

Patient-Directed Assessment & Intervention

Due to the potential for sudden deterioration and the insensitivity of symptoms in warning patients about the severity and variability in their asthma, an objective measure of airflow limitation is required for the optimal management of patients with asthma. Spirometry is not feasible for patients to perform in the home, but peak flow can be measured reliably and inexpensively. The technique requires a maximal exhalation into a hand-held measuring device. Many different brands of peak-flow meters are available. Reliability of the measuring device and reproducibility of measurements over time are probably more important than absolute accuracy of measurements.[8] Peak-flow monitoring is considered by clinical experts to be an integral part of care for patients with asthma. The NHLBI Guidelines indicate that peak flow should be monitored in patients with moderate, severe, or unstable asthma.[6] Peak-flow self-monitoring and acting upon the results as outlined by a healthcare practitioner are components of collaborative self-management in asthma. Despite the acceptance of the importance of peak-flow monitoring by expert clinicians, few investigations have rigorously evaluated the effects of peak-flow monitoring by patients. Most of the available studies have included peak flow only as part of an education and self-management program. Many of the studies enrolled limited numbers of subjects and not all studies employed control groups. Clark et al have recently reviewed the available literature on the use of peak-flow monitoring and note the need for further studies of the effectiveness of peak-flow monitoring.[9]

Charlton et al emphasize the need for education coupled with peak-flow measurements.[10] They randomized 115 children and adults with asthma in a rural nurse-run asthma clinic to receive self-management instruction based upon symptoms or self-management based upon peak-flow measurements. The authors attributed the lack of difference in outcomes between the two groups to the comprehensive nature and multiple components of the program that included care by a specially trained nurse, longer appointments, regular follow-up, review of inhalation technique, specific management plans, and education. Thus, the precise role of peak flow cannot be deduced from this study.

Janson-Bjerklie and Shnell found decreased medication use by patients who used peak-flow measurements compared to that of patients who relied on symptom assessment alone.[11] Patients using peak-flow meters also used more alternative non-medication treatment strategies, but other outcomes that might have assessed the appropriateness of self-management were not measured. It cannot be determined from this study whether the decreased medication use was associated with reduction in symptoms or improved airflow limitation. Of interest, none of the patients in this study used medications as originally prescribed; doses and frequency of medications were altered by patients without their physicians' knowledge.

Creer et al reported the effects of an 8-week program that included peak-flow monitoring in 123 children with asthma.[12] Patients were taught self-management skills using written materials during 4 group-training sessions, and peak flow was used as a guide to self-management. School absences were reduced in children from 17.5 days/year prior to the intervention to 11.0 days in the year after education and to 6.4 days in the second year following intervention. Direct expenditures, including medications and physician visits for childhood asthma, decreased 34%. Peak-flow measures improved over the course of the study indicating reduced airflow limitation. However, the role of peak-flow monitoring in achieving the demonstrated outcomes cannot be assessed in this study.

The available studies suggest that peak flow as a collaborative self-management tool in asthma may be associated with improved outcomes, including reduction in symptoms and improved lung function. Additional studies are clearly warranted to further define the role of peak flow and compare alternative strategies for self-management.

Several studies have investigated the efficacy of self-management programs in adults with asthma (Table 3).[10,11,13-20] These investigations document a reduction in the need for emergency care as a consequence of self-management.

Table 3. Reports of Collaborative Self-Management in Adults with Asthma

First Author	Results
Maiman[13]	Fewer emergency room visits
Hilton[14]	Increased asthma knowledge Improved satisfaction ? decreased emergency room visits
Janson-Bjerklie[11]	Decreased medication use
Beasley[15]	Decreased oral steroid use Improved symptoms Decreased airflow limitation Fewer work/school absences
Bailey[16]	Improved knowledge and medication skills Greater medication adherence Decreased symptoms
Charlton[10]	No effect
Mayo[17]	Fewer hospitalizations
Ringsberg[18]	Improved knowledge and metered-dose-inhaler technique Fewer hospitalizations and emergency room visits Improved quality of life
Wilson[19]	Decreased symptoms Improved MDI technique
Mason[20]	Improved quality of life Fewer hospitalizations and emergency room visits

The effects of an education program for asthmatics seen in the emergency room were reported in an early study by Maiman et al.[13] They randomized 254 subjects to receive individual education at the time of the emergency visit or subsequent written material in the form of an information booklet and follow-up reinforcement by telephone. The intervention that was most effective in reducing emergency room visits was individual instruction at the time of the emergency-room visit by a nurse educator. Instruction by an educator who was an asthmatic was more effective than instruction by one who did not have asthma. There was no benefit from the written information subsequently provided to the patient. Thus, these results do not support the generally accepted recommendation for written materials to enhance education.[6]

Mayo et al enrolled 104 asthmatics from a New York City hospital in a randomized trial of self-management.[17] Patients selected for the study had at least two hospitalizations in the previous 2 years or at least five emergency room visits in the previous year. Patients were randomized to receive routine care or instruction by a physician in addition to routine care. Instruction consisted of at least two 1-hour discussions of pathophysiology, treatment, and self-management strategies to meet each patient's needs. No written materials or formal lectures were provided. Treatment was simplified to include the least possible number of medications. Patients were encouraged to contact medical personnel whenever necessary outside of scheduled visits and to seek emergency care early when necessary. Peak-flow monitoring was included. Patients participated in management decisions, and "their preferences were respected." During the study, all self-management patients used metered-dose inhaler (MDI) bronchodilators in conjunction with spacers or holding chambers and peak-flow meters, and 89% used steroid pulses. Self-management patients had a significant reduction in hospitalizations, both when compared to admission rates prior to the study and when compared to control patients. The reduction in hospital admissions persisted for 4 years. However, the medical treatment of the self-management group changed during the course of the study. Prior to entering the study, none of the self-management patients had used outpatient oral corticosteroids, MDIs with holding chambers, or home peak-flow monitoring. The authors appropriately note that it is not clear which of the major components of care (physician availability, patient education, or medication delivery) were responsible for the beneficial outcomes.

The effects of providing asthmatics with a comprehensive collaborative self-management approach were investigated by Beasley et al.[15] A detailed collaborative self-management plan with specific instructions to modify medications based upon values of peak-flow was given to 30 consecutive outpatients. Medication adjustments included modification of inhaled steroid doses and use of oral steroid pulses. Over a 7-month average follow-up, patients demonstrated significant improvements: improved airflow as measured by spirometry (FEV_1 increase from 76% predicted before self-management to 92% predicted following self-management), decreased nocturnal awakenings (4.3 awakenings in 2 weeks decreased to 0.7), reduction in days lost from work or school (from 12.9 days in 6 months to 1.7 days), increased doses of inhaled corticosteroids, and decreased use of antibiotics and oral corticosteroids. Thus, this study documented that increased doses of inhaled corticosteroids and patient monitoring of peak flow decreased airflow limitation and oral steroid use. However, the lack of a control group makes it difficult to interpret the role of the other self-management components such as education and peak-flow monitoring compared to the increased use of inhaled steroids.

Group education is imparted to patients in Sweden through "asthma schools." Ringsberg et al[18] demonstrated that such education increased patient knowledge and improved inhaled medication technique compared to patients who were not enrolled in the asthma school. In addition, there was a reduction in emergency-room visits and improved quality of life. Although both groups had reduced hospitalizations over the year following intervention, patients in the asthma school had a greater reduction in hospital days. However, it appears that the study patients had more hospitalizations prior to intervention than control patients. Patients in this study had more severe disease and were enrolled in the study only if they had one or more hospitalizations in the previous year. It is unclear whether this program would have similar effects in patients with less severe disease.

Bailey et al studied the effects of an educational program integrating a workbook with a 1-hour counseling session by a health educator.[16] They randomized 267 patients to receive the education (self-management) program or usual care. Self-management patients had improved knowledge and medication skills. The education group also demonstrated greater adherence to medications and decreased asthma symptoms. Reductions in emergency department visits and hospitalizations were similar in both groups. The relative importance of the counseling and written materials was not investigated.

Two studies have reported much less substantial benefits following educational programs.[14,19] Office-based general practitioners in London, United Kingdom, enrolled 274 patients with asthma (ages 5-70 years) in a controlled study comparing two different types of education to a control group.[14] A "maximum" education group received a written booklet followed in 1 month by a treatment card, a 10- to 15-minute semistructured physician interview, an audiocassette tape, follow-up physician appointments every 3 months, and routine asthma care. A more limited education program (including only standard care, the information booklet, and treatment card) was evaluated in a second group, and a control group received no instruction. Knowledge improved in the maximum education group but there were no effects on self-management of asthma attacks, reported medication adherence, or inhaler technique. Likewise, there were no differences in lost work or school days, frequency of wheezing or asthma attacks, nocturnal awakenings, or physician home visits. The authors conclude that increased patient satisfaction and knowledge were "accompanied by" a reduction in emergency hospital visits but give no data to support this conclusion.

The effects of different types of education employed in a health maintenance organization were evaluated by Wilson et al.[19] They randomized 323 patients with asthma recruited from 5 medical centers to receive either group instruction or individualized one-on-one instruction delivered by a nurse educator. Two control groups included patients who received no instruction and patients who were given access to educational materials provided in a workbook. Both education programs resulted in patients, using a standardized questionnaire, noting they were significantly less "bothered" by their asthma, had fewer symptomatic days, and had improved technique for using MDIs despite no difference in the degree of airflow limitation as measured by spirometry. It is unclear whether the decreased symptoms noted were related to improved MDI technique. The small group education program resulted in an increase in physical activity and clinical improvement based on physician assessment. Access to medical information in the form of the workbook alone did not improve any of the above parameters. There was no change in hospitalizations or in acute physician visits in the year following education. Patients were considered by their physicians to have moderate to severe asthma if they had required ≥ 3 physician visits for asthma in the year prior to the study and had received daily medication for their disease. Nevertheless, only 8% of patients were hospitalized for asthma in the year prior to the study, suggesting this population was less severely affected than patients enrolled in other studies.

At the National Jewish Center for Immunology and Respiratory Medicine, collaborative self-management has resulted in improved quality of life and reduced healthcare utilization.[20] Patients with asthma spend 1 week at the Center for evaluation and education. The educational focus is on informing patients about their illness and its management and actively engaging them in collaborative self-management. Group and individual sessions also include instruction on medication technique and administration. Peak-flow monitoring by patients is routinely employed. A recent pilot study randomly selected 48 patients for longitudinal assessment of quality of life and healthcare utilization outcomes. The self-administered Medical Outcome Study 36-Item Short-Form Health Survey (SF-36) designed by Dr John Ware[21] was used as a measure of quality of life before the program and at 1, 6, and 12 months after the program. Six of the quality-of-life scales (including physical function, physical role, social function, emotional role, energy/fatigue, and perception of health) improved significantly at 1 month and appear to remain improved for 1 year (Fig. 1). In addition, dyspnea and perceived interference with work, derived from the quality-of-life scales, decreased (Fig. 2). Hospital days decreased by 82% (Fig. 3) and emergency room visits decreased by 45% (Fig. 4) in the year following intervention. All but one of the 13 patients who had at least 2 hospital days in the prior year showed a reduction in hospital days in the year after intervention.

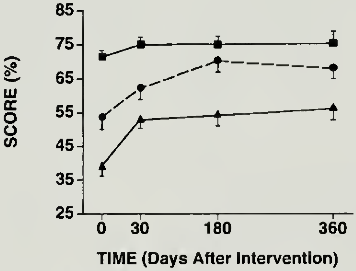

Fig. 1. Mean (SE) scores of mental health (squares), physical subscale (circles), and energy/fatigue subscale (triangles) of the Medical Outcomes Study Health Status Index in 48 patients with asthma before and at 30, 180, and 360 days after participation in the National Jewish Center program. Higher scores indicate improved patient perception of quality of life. (Reprinted from Reference 20.)

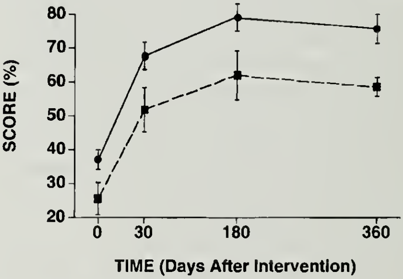

Fig. 2. Mean (SE) scores of health change subscale (circles) and role-physical subscale (squares) of the Medical Outcomes Study Health Status Index in 48 patients with asthma before and at 30, 180, and 360 days after participation in the National Jewish Center program. Higher scores indicate improved patient perception of quality of life. (Reprinted from Reference 20.)

Collaborative Self-Management in Children with Asthma

Numerous studies have evaluated the outcomes of asthma self-management programs, and these studies have been summarized in recent reviews.[22-24] Self-management in children has been shown to reduce symptoms such as wheezing,[25,26] decrease uti-

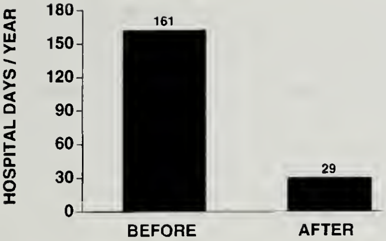

Fig. 3. Total number of hospital days/year for 48 patients before and after the National Jewish Center asthma program. (Reprinted from Reference 20.)

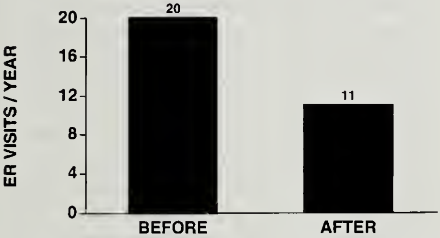

Fig. 4. Total number of emergency room visits/year for 48 patients before and after the National Jewish Center asthma program. (Reprinted from Reference 20.)

lization of acute healthcare resources including emergency rooms and hospitals,[27-31] decrease school absences and improve grades,[25,26,29,32] increase physical activity,[28] and decrease anxiety.[29,33] However, many of these studies used inadequate study designs, enrolled too few patients, or failed to test hypotheses using statistical methods. Nonetheless, the published investigations indicate that involvement of asthmatic children and their families in their own care can improve outcomes, most notably decrease absences from school and reduce the frequency of emergency-room visits and hospitalizations (Table 4).

One of studies that comprehensively investigated the effects of self-management in children with asthma was reported by Clark et al.[29] They randomized 310 low-income urban children with asthma to participate in self-management or to receive standard care. The self-management group had fewer hospital admissions and emergency visits, better grades in school, and less anxiety about their illness. The children enrolled in the study were between 4 and 17 years of age and had reported one or more episodes of wheezing in the previous year. Children were not enrolled on the basis of their prior need for emergency medical care. Patients and families in the self-management group attended six 1-hour

Table 4. Reports of Collaborative Self-Management in Children with Asthma

First Author	Results
Studies Showing Significant Changes	
Creer[25]	Decreased wheezing Decreased school absence
Parcel[33]	Greater feeling of control Reduced anxiety
Fireman[32]	Decreased school absence
Hindi-Alexander[28]	Decreased school absence Increased asthma knowledge Increased physical activity
Lewis[27]	Increased knowledge Fewer emergency room and hospital visits
Wilson-Pessano[34]	Increased self-management
Rubin[30]	Fewer emergency room and hospital visits
Clark[29]	Increased self-management Fewer emergency room and hospital visits Improved school grades Reduced anxiety
Evans[26]	Increased self-management Improved school grades Fewer asthma episodes
Studies Showing Change of Unspecified Significance	
Blumenthal[35]	Decreased school absence
Robinson[36]	Decreased school absence
Kohen[37]	Decreased school absence Fewer emergency room visits
Creer[12]	Decreased school absence Decreased costs associated with care
Studies Showing No Change	
Staudenmayer[38]	
Rakos[39]	

group classes that met monthly at the time of the scheduled clinic visit. Classes were offered in both English and Spanish, and the discussion topics included managing an asthma attack, medication administration, physician communication, school performance, home environment, and physical activity. Physician care was not changed during the study, and physicians did not know which patients were receiving instruction. Children and families participating in self-management education increased their involvement in their care. Patients who had no hospitalizations in the year prior to the study had no change in hospital admissions or emergency room visits in the year following intervention. In the 35 children who were hospitalized in the year prior to the study, patients in the self-management group had a decrease in hospitalizations and more than a 50% reduction in emergency room visits compared to control patients. Education of 19 high-resource users (those with one or more hospitalizations prior to the study) saved a total of $17,481 due to reduced hospital and emergency room utilization at a cost of only $1,558 for the educational program. This study clearly demonstrates the benefits of education to children with asthma and their families, even when the instruction is not provided by a physician. However, favorable outcomes were seen only in patients who had more severe asthma as indicated by the need for hospital and emergency room care prior to institution of self-management.

Hindi-Alexander and Cropp evaluated the effects of a 6-week, 12-hour education program on 147 children with asthma.[28] Both children and parents increased their knowledge of asthma and participation in activities in and out of school improved. Unscheduled healthcare visits decreased by 51% and school absences decreased by 48%. The number of episodes of wheezing demonstrated a nonsignificant increase. Thus, although the patients' asthma was not necessarily better controlled, self-management had an effect as shown by the decreased need for medical assistance and increased ability to participate in school and activities. These results have been duplicated in other studies. The ability of patients and families to learn and apply self-management techniques was also demonstrated by Evans et al[26] and Wilson-Pessano and Webb.[34]

The available studies in children indicate that collaborative self-management is most effective for patients with more severe asthma, particularly patients who have required emergency room visits and hospitalizations prior to its institution. However, it should be recognized that more global indicators of quality of life have not been measured in children, and only a few studies have assessed asthma symptoms or degree of airflow limitation. Additional studies assessing these indexes in response to comanagement are clearly indicated.

Collaborative Self-Management in Patients with Chronic Obstructive Pulmonary Disease

Little information has been published on self-management in patients with chronic obstructive pulmonary disease (COPD). Because many of the medications used by these patients and the principles of care are similar to asthma, the concepts discussed for asthma management may be similar for COPD.

Patients with COPD may benefit from collaborative self-management related to their use of medications. For example, a recent study by Dolce et al documented suboptimal medication use in 35 patients with COPD.[40] Seventy-eight outpatients were instructed by their physicians to use an average of 6.3 medications per patient. On average, patients regularly used 2.3 oral medications and 60% of patients were instructed to use 2 or more regular doses of inhaled bronchodilators. Adherence was poor; 54% of patients underutilized their medications because they either forgot to take them or because of a conscious decision not to use them. Half of the patients overused their medications during periods of increased symptoms. Metered-dose inhaler technique was also ineffective in 24% of patients.

The goal of pulmonary rehabilitation for patients with COPD is to improve the quality of life. Education is one of the key components of pulmonary rehabilitation[41-43] and thus it should be expected that outcomes would be improved following a pulmonary rehabilitation programs that incorporates education. While it is generally accepted that exercise training in COPD improves subsequent exercise performance, the addition of an educational component in the context of collaborative self-management might be expected to improve quality of life. A brief description of the National Jewish Center Pulmonary Rehabilitation Program highlights the outcomes that might be anticipated following pulmonary rehabilitation. We have investigated physiologic, functional, and quality-of-life outcomes following a 3-week rehabilitation program that incorporated exercise training to improve physical endurance and group and individualized instruction to enhance collaborative self-management. In 26 consecutive patients with moderately severe COPD ($FEV_1 = 48\%$ predicted), the average maximum work-load increase was 30% and the average maximum oxygen consumption increase was 9.2%.[44] Maximum heart rate was unchanged following training, suggesting that a physiologic training effect was achieved. In another study of 21 consecutive patients, quality of life and functional capacity increased.[45] Six-minute walk distance was used as a functional measure and was found to improve by 21%. Using the self-administered Medical Outcome Study 36-Item Short-Form Health Survey,[21] quality of life was also found to improve. Following rehabilitation, significant improvements were seen in the scales used to assess patient perception of physical function, social function, emotional role, mental health, energy/fatigue and health change. Studies of pulmonary rehabilitation generally employ multiple therapeutic modalities to improve outcomes, and thus the precise role of each of the individual components is unclear.

It is generally accepted that the goal of education for patients should be to elicit a behavioral change rather than solely to provide theoretical knowledge.[46,47] The role of a behavioral component has been investigated in patients with COPD.[48-50] A San Diego group evaluated the exercise and quality-of-life outcomes of a behavioral program in 76 patients with moderate to severe COPD.[48,49] Patients were randomized to one of five interventions: behavior modification, cognitive modification, cognitive-behavior modification, attention control, or no-treatment control. Treatment-group patients met individually with a health professional 7 times over 3 months. The first and last sessions were held in the health-care setting, and five sessions were conducted at the patient's home. Behavior modification included a signed contract with the patient who agreed to set specific times for walking exercise, training in muscle relaxation, and breathing retraining. Individuals in the cognitive-modification group were trained to replace negative thoughts, feelings, and behaviors with more positive ones. The attention-control group spent time with a counselor and was given information about managing stressful life events and about family support. The time patients spent in walking exercise and quality of life were increased in the 3 treatment groups. In addition, patients in the cognitive-behavior group did better than those in the attention-control group. Thus, an educational program, that included specific interventions to modify behavior, improved outcomes.

The results of a community-based study of the effect of education on health outcomes and perceptions of 538 patients with COPD was reported by Howland et al.[50] Patients with COPD in one community were offered an education program consisting of six 2-hour sessions emphasizing smoking cessation, stress reduction, and building physical endurance through exercise. Patients in a nearby community were used as controls. Outcome variables measured by questionnaires included respiratory symptoms, health perception, and locus of control (whether patients consider health status under their own control), mental health, physical function, and social function. Patients in the education group increased their locus of control, but there were no effects on symptoms or quality of life. The authors suggest that an education program alone has limited benefits for patients with COPD.

Many patients with COPD seem most concerned about one particular aspect of their treatment—the chronic use of supplemental oxygen. In carefully controlled studies of the efficacy of ambulatory oxygen therapy, it appears that patients use oxygen less than prescribed. The Nocturnal Oxygen Therapy Trial compared use of nocturnal oxygen with use of continuous oxygen prescribed for 24 hours a day.[51] Although patients were provided with instruction and monitoring in this multicenter trial, the continuous-oxygen group used oxygen for 17.7 hours per day. Patients used various delivery systems, including cylinders of compressed gas and liquid oxygen both via stationary and portable systems. The relative lack of portability of bulky compressed gas systems may have impaired the ability of some patients to use oxygen more consistently. In addition, the use of oxygen was monitored only when the stationary system was used; thus, measured oxygen use may have underestimated actual use. Although it appears that oxygen is underused by patients, there is little information available assessing the role of collaborative self-management for COPD patients who use ambulatory oxygen. Respiratory care practitioners (RCPs) who visit the home to assess the use of oxygen therapy are the most appropriately positioned healthcare professionals to encourage collaborative self-management of oxygen therapy. RCPs should reinforce the use of oxygen through education with the goal of increasing the appropriate use and dosing of oxygen therapy at rest, during exercise, and during sleep.

Other Applications for Collaborative Self-Management

Three other areas of respiratory home care (continuous positive airway pressure for sleep-disordered breathing, mechanical ventilation, and smoking cessation) should be considered for collaborative self-management. Although little information has been published on collaborative self-management in the first two of the conditions, patients who require these interventions share the need for involvement in their own care.

Sleep-Disordered Breathing

Continuous positive airway pressure (CPAP) is frequently prescribed for individuals with sleep-related breathing disorders. However, patient adherence to use of CPAP is less than optimal. One recent study found that patient reports of CPAP use was 6.1 hours per night while actual measured use was only 4.9 hours per night.[52] Collaborative self-management might be expected to improve patient adherence with the use of CPAP. However, further studies to evaluate the effectiveness of co-management in patients using CPAP are necessary.

Home Mechanical Ventilation

Little information has been published on home ventilation. Our experience, however, is that these technology-dependent patients have a strong desire to be involved in their own care. Patients with lifelong neuromuscular and skeletal disorders, just as in most patients with other chronic disabling diseases that affect quality of life, wish to be in control of their lives and their futures. Patients who are disabled due to chronic neuromuscular disorders and unable to care for themselves may nonetheless be able to direct the care provided by others such as paid attendants. Self-management and education may improve the ability of patients and caregivers to prevent, detect, and correct problems that occur in the home.

The National Center for Home Ventilation was founded in 1990 to improve the safety and efficacy of home ventilation. The Center has developed a registry of over 450 patients receiving positive pressure ventilation in the home and continues to acquire new patients and expand its database. In addition to the registry, RCPs in the home are asked to inform the Center of patient encounters when (1) phone calls are received from patients, healthcare professionals, or caregivers reporting a new clinical situation, suspected equipment malfunction, or suspected supply problem; (2) unscheduled visits are performed by RCPs; and (3) supplies or equipment fail to function properly, a ventilation system component is improperly assembled, or equipment is not employed as prescribed. In tracking encounters between patients and RCPs, unscheduled home visits by RCPs for suspected problems are frequently resolved with education of the patient and caregivers.

The Center has received 11 contact reports relating to humidifiers, 10 related to alarms, 9 related to circuits, and 8 related to tracheostomies. RCPs may have reported more than one problem/solution related to each contact. In addition to attending to mechanical problems, RCPs documented re-instruction as an appropriate solution for 6 of these. Issues related to alarms, circuits, and tracheostomies were also frequently resolved with re-instruction.

Even when patients are educated in the hospital prior to discharge and at the time of discharge to the home, they may not recall all the information provided. As an aid to healthcare professionals who manage ventilator-assisted individuals in the home, the Center has developed a list of potential educational objectives to guide education of the patient and caregivers.[53] The educational process for technology-dependent patients in the home should include identification of the learning objectives; education of patients and caregivers using oral, visual, and written methods; evaluation of the information learned; and periodic reinforcement. Because of their expertise in this area and their involvement in both hospital and home care, RCPs are one of the keys to assuring that collaborative self-management of technology-dependent patients with respiratory disorders in the home is successful.

Smoking Cessation

Smoking cessation can be effective only when the patient is ready to quit smoking—a classic example of collaborative self-management. Contracting with the patient to stop smoking has been advocated as a method of including the patient in his own care. Although physician advice is a strong motivator in achieving smoking cessation, patients must be receptive to the advice and willing to act accordingly. RCPs are often in the position to initiate and facilitate smoking cessation, not only with hospitalized patients but also with patients receiving home respiratory care and parents of children with respiratory disease.[54] However, as noted in a recent editorial in this journal, RCPs may be smokers themselves and, as such, may not the best role models for their patients.[55] The American Association for Respiratory Care has developed materials (the Nicotine Intervention Kit) to assist RCPs in playing an active role in smoking cessation in the hospital setting. These interventions should be extended to the home setting.

Summary

The effectiveness of collaborative self-management of respiratory disorders has been most clearly demonstrated in asthma. In both adults and children with severe asthma requiring emergency care and hospitalizations, collaborative self-management can decrease not only the need for emergency care and hospitalization but also time lost from work and school, thereby increasing the patients' ability to be full and active participants in the community. Collaborative self-management is best provided in a comprehensive program that includes ready access to healthcare professionals, education, behavioral therapy, and peak-flow monitoring. The relative value of each of these components and the value of single components applied simultaneously is unclear.

In some studies, asthma symptoms and medication adherence have also improved following implementation of collaborative self-management. The available data indicate that collaborative self-management should be considered as a potential therapeutic adjunct in the management of every patient with asthma and should be routinely employed with patients who have severe disease as defined by emergency room use and hospitalizations. Additional investigations should be conducted in patients with other respiratory diseases to define the role of collaborative self-management. Nonetheless, state-of-the-art medical care and the nature of the patient-physician relationship in the 1990s dictates that collaborative self-management be routinely employed in the optimal outpatient management of any individual who is interested in participating in his or her own healthcare. However, individualization of the practitioner-patient relationship is necessary. Not all patients desire to play an active role in their illness and its management. The personality, attitudes, and desires of individuals in assisting in their own management must be assessed. Theory would suggest that patients who do not appear to have an interest in self-management may have low self-efficacy that may be increased by educational efforts. Improved self-efficacy and active participation in disease management may result in beneficial outcomes. It is clear that the physician-patient relationship has changed over the past decade. As patients have been empowered to act in their own best interests, the physician's role as an educator and facilitator has become more prominent than ever. Even if medical investigations had not demonstrated any beneficial effects of collaborative self-management, patients have the right to participate in decisions affecting their healthcare and are increasingly exercising that right. Respiratory care practitioners are in a unique position to enhance collaborative self-management. In the home environment, RCPs can foster smoking cessation and serve as a liaison between patients and physicians. Education and reinforcement of collaborative self-management should be a part of every home visit performed by respiratory care practitioners, particularly in patients using CPAP, oxygen, and mechanical ventilation.

In 1993 it seems unnecessary to suggest that healthcare practitioners should, as a National Institutes of Health Workshop recommended in 1989, "encourage education as a regular part of routine asthma care."[56] Our patients are far ahead of this simple recommendation and are demanding more of their healthcare practitioners. We need to be prepared to institute collaborative self-management in all patients with respiratory disease.

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REFERENCES

1. Bartlett EE. Educational self-help approaches in childhood asthma. J Allergy Clin Immunol 1983;75(5, Part 2):545-554.
2. Hindi-Alexander MC, Throm J, Middleton E Jr. Collaborative asthma self-management: evaluation designs. Clin Rev Allergy 1987;5(3):249-258.
3. Wilson-Pessano SR, Mellins RB. Workshop on asthma self-management: summary of workshop discussion. J Allergy Clin Immunol 1987;80:487-490.
4. Bandura A. Social learning theory. Englewood Cliffs NJ: Prentice-Hall, 1977.
5. American Thoracic Society. Withholding and withdrawing life-sustaining therapy. Am Rev Respir Dis 1991; 144(3, Part 1):726-731.
6. National Heart, Lung & Blood Institute. Expert panel report: guidelines for the diagnosis and management of asthma. Bethesda MD: NHLBI, 1991.
7. Reinke LF, Hoffman LA. Breathing space: how to teach asthma co-management. Am J Nurs 1992;92(10):40-46.
8. Simmons M, Wynegar T, Hess D. Evaluation of the agreement between portable peak flow meters and a calibrated pneumotachometer. Respir Care 1993;38(8):916-922.
9. Clark NM, Evans D, Mellins RB. Patient use of peak flow monitoring. Am Rev Respir Dis 1992;145(3):722-725.
10. Charlton I, Charlton G, Broomfield J, Mullee MA. Evaluation of peak flow and symptoms only self management plans for control of asthma in general practice. BMJ 1990;301(6765):1355-1359.
11. Janson-Bjerklie S, Shnell S. Effect of peak flow information on patterns of self-care in adult asthma. Heart Lung 1988;17(5):543-549.
12. Creer TL, Backial M, Burns KL, Leung P, Marion RJ, Miklich DR, et al. Living with asthma. I. genesis and development of a self-management program for childhood asthma. J Asthma 1988;25(6):335-362.
13. Maiman LA, Green LW, Gibson G, MacKenzie EJ. Education for self-treatment by adult asthmatics. JAMA 1979;241(8):1919-1922.
14. Hilton S, Sibbald B, Anderson HR, Freeling P. Controlled evaluation of the effects of patient education on asthma morbidity in general practice. Lancet 1986; 1(8471):26-29.
15. Beasley R, Cushley M, Holgate ST. A self management plan in the treatment of adult asthma. Thorax 1989; 44(3):200-204.
16. Bailey WC, Richards JM Jr, Brooks CM, Soong SJ, Windsor RA, Manzella BA. A randomized trial to improve self-management practices of adults with asthma. Arch Intern Med 1990;150(8):1664-1668.
17. Mayo PH, Richman J, Harris HW. Results of a program to reduce admissions for adult asthma. Ann Intern Med 1990;112(11):864-871.
18. Ringsberg KC, Wiklund I, Wilhelmsen L. Education of adult patients at an "asthma school": effects on quality of life, knowledge and need for nursing. Eur Respir J 1990;3(1):33-37.
19. Wilson SR, Scamagas P, German DF, Hughes GW, Lulla S, Coss S, et al. A controlled trial of two forms of self-management education for adults with asthma. Am J Med 1993;94(6):564-576.
20. Mason RJ, Katz JL, Bethel RA. Time out for asthma: rationale for a comprehensive evaluation. Semin Respir Med 1994 (in press).
21. Stewart AL, Hays RD, Ware JE Jr. The MOS short-form general health survey: reliability and validity in a patient population. Med Care 1988;26(7):724-735.
22. Howland J, Bauchner H, Adair R. The impact of pediatric asthma education on morbidity: assessing the evidence. Chest 1988;94(5):964-969.
23. Klingelhofer EL, Gershwin ME. Asthma self-management programs: premises, not promises. J Asthma 1988; 25(2):89-101.
24. Clark NM, Gotsch A, Rosenstock IR. Patient, professional, and public education on behavioral aspects of asthma: a review of strategies for change and needed research. J Asthma 1993;30(4):241-255.
25. Creer TL, Renne CM, Christian WP. Behavioral contributions to rehabilitation and childhood asthma. Rehabil Lit 1976;37(8):226-232,247.
26. Evans D, Clark NM, Feldman CH, Rips J, Kaplan D, Levison MJ, et al. A school health education program for children with asthma aged 8-11 years. Health Educ Q 1987;14(3):267-279.
27. Lewis CE, Rachelefsky G, Lewis MA, de la Sota A, Kaplan M. A randomized trial of ACT (asthma care training) for kids. Pediatrics 1984;74(4):478-486.
28. Hindi-Alexander MC, Cropp GJA. Evaluation of a family asthma program. J Allergy Clin Immunol 1984;74(4, Part 1):505-510.
29. Clark NM, Feldman CH, Evans D, Levison MJ, Wasilewski Y, Mellins RB. The impact of health education on frequncy and cost of health care use by low income children with asthma. J Allergy Clin Immunol 1986;78(1, Part 1):108-115.
30. Rubin DH, Leventhal JM, Sadock RT, Letovsky E, Schottland P, Clemente I, et al. Educational intervention by computer in childhood asthma: a randomized clinical trial testing the use of a new teaching intervention in childhood asthma. Pediatrics 1986;77(1):1-10.

31. Detwiler DA, Boston LM, Verhulst SJ. Evaluation of an educational program for asthmatic children ages 4-8 and their parents. Respir Care 1994;39(3):204-212.
32. Fireman P, Friday GA, Gira C, Vierthaler WA, Michaels L. Teaching self-management skills to asthmatic children and their parents in an ambulatory care setting. Pediatrics 1981;68(3):341-348.
33. Parcel GS, Nader PR. Evaluation of a pilot school health education program for asthmatic children. J Sch Health 1977;47(8):453-456.
34. Wilson-Pessano SR, McNabb WL. The role of patient education in the management of childhood asthma. Prev Med 1985;14(6):670-687.
35. Blumenthal MN, Cushing RT, Fashingbauer TJ. A community program for the management of bronchial asthma. Ann Allergy 1972;30(7):391-398.
36. Robinson LD Jr. Evaluation of an asthma summer camp program. Chest 1985;87(1, Suppl):105s-107s.
37. Kohen MD. Educational and exercise programs for asthmatic children. South Med J 1985;78(3):948-950.
38. Staudenmayer H, Harris PS, Selner JC. Evaluation of a self-help education-exercise program for asthmatic children and their parents: six-month follow-up. J Asthma 1981;18(1):1-5.
39. Rakos RF, Grodek MV, Mack KK. The impact of a self-administered behavioral intervention program on pediatric asthma. J Psychosom Res 1985;29(1):101-108.
40. Dolce JJ, Crisp C, Manzella B, Richards JM, Hardin JM, Bailey WC. Medication adherence patterns in chronic obstructive pulmonary disease. Chest 1991;99(4):837-841.
41. American Thoracic Society, Medical Section of the American Lung Association. Pulmonary rehabilitation (ATS statement). Am Rev Respir Dis 1981;124(5):663-666.
42. Make BJ. Pulmonary rehabilitation: myth or reality? Clin Chest Med 1986;7(4):519-540.
43. Ries AL. Position paper of the AACPR. Scientific basis of pulmonary rehabilitation. J Cardiopulmonary Rehabil 1990;10:418-441.
44. Make BJ, Buchholz J. Exercise training in COPD patients improves cardiac function (abstract). Am Rev Respir Dis 1991;143(4, Part 2):A80.
45. Make B, Glenn K, Iklé D, Bucher B, Tuteur D, Mason U, et al. Pulmonary rehabilitation improves the quality of life of patients with chronic obstructive pulmonary disease (COPD) (abstract). Am Rev Respir Dis 1992;145(4, Part 2):A767.
46. Windsor RA, Green LW, Roseman JM. Health promotion and maintenance for patients with chornic obstructive pulmonary disease: a review. J Chronic Dis 1980;33(1):5-12.
47. Mazzuca SA. Does patient education in chronic disease have therapeutic value? J Chronic Dis 1982;35(7):521-529.
48. Toevs CD, Kaplan RM, Atkins CJ. The costs and effects of behavioral programs in chronic obstructive pulmonary disease. Med Care 1984;22(12):1088-1100.
49. Atkins CJ, Kaplan RM, Timms RM, Reinsch S, Lofback K. Behavioral exercise programs in the management of chronic obstructive pulmonary disease. J Consult Clin Psychol 1984;52(4):591-603.
50. Howland J, Nelson EC, Barlow PB, McHugo G, Meier FA, Brent P, et al. Chronic obstructive airway disease: impact of health education. Chest 1986;90(2):233-238.
51. Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Ann Intern Med 1980;93(3):391-398.
52. Rauscher H, Formanek D, Zwick H. Self-reported vs measured compliance with nasal CPAP for obstructive sleep apnea. Chest 1993;103(6):1675-1680.
53. Glenn K, Make B. Learning objectives for positive pressure ventilation. National Center for Home Mechanical Ventilation. 1993.
54. Nett L. Why we must help parents stop smoking (editorial). Respir Care 1993;38(9):982-984.
55. Robbins YJ, Albright DC, Deutsch ET Jr. Walking the walk—not just talking the talk: cigarette smoking and respiratory care practitioners (editorial). Respir Care 1993;38(10):1069-1071.
56. Parker SR, Mellins RB, Sogn DD. NHLBI Workshop Summary. Asthma education: a national strategy. Am Rev Respir Dis 1989;140(3):848-853.

Make Discussion

Rodriguez: The current JCAHO standards for hospitals place great emphasis on patient and family education, and on the development of self-care skills learning programs. Have similar standards been established for patient and family education in the home?

Make: I don't know. Maybe somebody can comment on that.

Dunne: Yes, they have. As a matter of fact, the emphasis of the 1995 standards will be a direct focus on patient education that follows the development of the plan of service or care that is based on an initial assessment. The reformatting of the standards for home care and for acute care, I might add, is intended to draw attention to the key processes that affect the outcome of care. Clearly, an initial assessment to gather relevant data to facilitate the formulation of a realistic plan of care of service, including educational outcomes, is of paramount importance. Thus, the education and training processes are identified as discrete key processes within themselves. They will have their own scoring guidelines and their own criteria for evaluation by the surveyor—because it's basically where the 'rubber hits the road,' as Sam (Giordano) likes to remind us. The follow-up, redemonstration, and the re-education become equally important. The data presented on home mechanical ventilation bore this fact out very, very clearly. If we only show a caregiver once or twice how to use a home mechanical ventilator with a humidification system, it is very obvious that problems could arise.

Mathews: One of the issues that I have been looking at for a long time is the quality of the educational materials, the printed materials, that we give to patients. We published a study in RESPIRATORY CARE[1] on the readability of patient education materials in which we found that the average readability of the samples that we picked was 11.8 years of education, whereas the educational norm for citizens of the United States is about 7.5. So there's a marked disparity. We went back and kind of informally looked at this again last year. I was talking to the people in the Kansas City area who are producing these materials, and there's been very little progress. It's down to a grade level of about 11.5 now. I would suggest that one thing we need to do when we're assessing patients is to assess their educational levels and assess their ability to read the materials we're giving to them. There is effective illiteracy in a large proportion of our population. People may speak well and present well, but they may not be able to read the materials that we give them.

REFERENCE

1. Mathews PJ, Thornton L, McLean L. Reading-level scores of patient education materials and the effect of teaching special vocabulary. Respir Care 1988;33(4):245-250.

Kacmarek: I think the mistake that's been made too often in training patients who are to go home with some type of mechanical ventilatory support is to rely on them to read printed literature. I believe you have to interact with the patient the same way I interact with practitioners in training them to use a piece of equipment. I want the patient or the caregiver to demonstrate to me specifically what I taught them—show me that they can actually perform the task, that they can turn the dials, that they can tell me what everything does or is going to do. I agree with you! How many of us read everything that's provided to us?

Mathews: The suggestions that we came up with for looking at the patient education literature was that you want to use pictures wherever you can and to use 1- to 2- or 3-word phrases that are very simple but convey important information.

Make: Patients need to learn the material first and then apply the information they have learned. Both cognitive knowledge and performance skills are important. Most of the studies on asthma have shown that patients can increase their knowledge. Written and verbal information must be conveyed in a language and at a readability level the patient can understand. Pictorial presentations are not optimal for all patients. There are people who don't learn visually—who are not very good at pictures. I agree that the educator must assess the patient's learning style and individualize the educational process.

Mathews: As educators we need to match the presentation with the learning style of the individual. We don't really do that. Another important factor is to put a glossary up front and introduce a new term in the beginning. Don't wait until the end and put in an appendix. It doesn't work. They don't look that far ahead.

Hoffman: You mentioned outcomes when using peak flow meters. One of my graduates, Lynn Reinke RN MSN, randomized about 40 patients with asthma to use of peak flow monitoring or care without this device.[1] None of the patients had a history of frequent exacerbations. She found no difference between the groups in bronchodilator use, episodes of chest tightness or wheezing, or selection of self-care strategies. However, the subjects who used peak flow monitoring felt they were more "in control" of their asthma. This suggests there may be benefits associated with peak flow monitoring—psychological factors—that translate into measurable outcomes in healthcare.

REFERENCE

1. Reinke LF, Hoffman LA, Zullo TG, Kettering DL, Fiehler PC. Effect of peak flow monitoring (PFM) on selection of self-care strategies and medication use in adults with asthma (abstract). Am Rev Respir Dis 1992;145 (4, Part 2):A476.

Hill: First, I'd like to underline your comments about the need for flexibility. Clearly, a lot of patients aren't ready for political correctness, put physicians or practitioners up on a pedestal, and are reluctant to take any kind of leadership role in their care. They want to be led. We have to encourage them to collaborate as much as possible, but they're still out there. Of course, there are others who don't want to hear anything and want to lead the whole show. We have to feel them out and let them believe that they are leading the show and try to nudge them in directions we think are more beneficial to them. The practitioner may have to be on top in some relationships, on the bottom in others, and hopefully try to end up somewhere in the middle in most. My second comment relates to the discussion about peak flow meters; I have a completely different experience. I am beginning to think that they're often worthless, particularly for long-term use. I hand them out to most of my asthmatics now. They come back after the first month or two with numbers plotted all over their data sheets, and they're kind of into it, initially. But after maybe 3-6 months, they tire of doing it and ask, "Why am I doing this? It's not helping me!" It's only in a few patients who have a major anxiety component with their asthma, who have trouble deciding when it's their airway disease and when it's their anxiety that's making them feel uncomfortable that I find it helpful. I can't get most of my patients to comply with peak flow meter use long term.

Make: That is a very appropriate comment. Janson-Bjerklie[1] found that asthmatics given peak flow meters actually used less medication. That may be in part because knowledge of peak flow reduced their anxiety. The investigators could not determine whether the reduced utilization of medication was appropriate. I find peak flow meters most helpful over a relatively short term after you initially evaluate the patient. You give the patient a meter and have them use it over a period of a week as you re-adjust the medications. It gives them an objective index of the status of their disease. Thereafter they use it for very specific reasons. Do they have morning dips that they're not recognizing? Are there times of the day that are worse, although they don't think they are? Is their peak flow as bad as their symptoms suggest? Long term, I think there are those asthmatics who will benefit from monitoring of peak flow also. Those asthmatics who may benefit the most are those who suddenly get into trouble, who experience repeated hospitalizations from sudden attacks. Those patients you might want to monitor very rigorously over a long period of time. But that's the minority of asthmatics.

REFERENCE

1. Janson-Bjerklie S, Schnell S. Effect of peak flow information on patterns of self-care in adult asthma. Heart Lung 1988;17(5):543-549.

Celli: I have a comment that I think deals with future direction. I loved your slide where you said that part of the problem is feeding back into the health system by the patients because over 14 or 15 hours a day they can't reach the health deliverer. Dr Robert Freedman in Boston has a computerized system for blood pressure that he has already presented, where patients can access a computer with a telephone. It is a press-a-button kind of system, and if x-number of buttons are pressed, responding to specific questions, a flag is raised, and the message goes out to the emergency room, and a health practitioner gets back to the patient. It seems to me, and we've been talking about that, that this would be a superb use of a technique for COPD patients or patients on ventilators by which they could access the health system without a person having to be at the other end of the phone 24 hours a day. It is only when a certain level is triggered that the health system responds to that call. I think it would be very interesting to expand our use of this potentially powerful tool.

Make: One group has reported use of a computer-assisted educational program.[1] I am not aware of any literature about the use of computers at home in patients with lung disease. My bias is that if a patient is going to be able to use a computer, they're more intelligent, probably more involved in their own care, and I sort of wonder whether patients who are at that level are going to need a computer-assisted management program.

REFERENCE

1. Rubin DH, Leventhal JM, Sadock RT, Letovsky E, Schottland P, Clemente I. Educational intervention by computer in childhood asthma: a randomized clinical trial testing the use of a new teaching intervention in childhood asthma. Pediatrics 1986;77(1):1-10.

Celli: He doesn't need a computer, he needs a telephone. The instructions are very simple. The computer is located as a receptor. The patient picks up the phone, dials a number. The computer responds with a voice message and begins a series of questions. The answers are usually 1 or 2—yes or no, yes or no, yes or no. You structure a plan by which the response could be then graded in terms of severity. You could close that loop that I think we're failing to close because nobody can be by the phone 24 hours a day.

Stoller: I have a question about subjective reporting as a valid endpoint. As regards collaborative self-management, should we insist on research-quality data for subjective reporting or can one reliably depend on the patient's saying, "Yeah, you know, I feel better with my peak flow meter." In other words, in a research context, one would insist upon some validated subjective status measurement. On a day-to-day clinical basis, what should we do?

Make: On a research basis, we should assure that we objectively measure a patient's perceptions of quality of life. However, few practitioners use these measures clinically on a regular basis. We're investigating whether routine quality of life measurements are clinically helpful. But, I think a single global question (asking the patient how he is doing) may be as clinically useful as a formal questionnaire. But, as of yet, we don't have sufficient information to compare that global question to a more formal tool.

Stoller: In clinical issues where there really are no compelling data about the efficacy of a treatment, should we simply be administering that treatment and depending upon the patient's global self-assessment or should we be insisting on more formal assessment of the efficacy of an unproven treatment?

Make: I think that trials of the clinical efficacy of a particular therapy should include formal measures of quality of life as one of the outcomes.

Bach: Barry, considering your earlier comment about patients' assessing their own quality of life, Campbell[1] showed that the global question—asking the patient if he is satisfied with life in general—is very valid and accurately reflects the patient's satisfaction with specific life domains such as family life, home environment, social life, and education. The patient's perception of his life satisfaction and quality of life is much more valid than someone else's trying to assess it. So, if you want to know how the patient feels, ask him, "How satisfied are you with your life?"

REFERENCE

1. Campbell A, Converse PE, Rodgers WL. The quality of American life: perceptions, evaluations, and satisfactions. New York: Russell Sage Foundation, 1976:37-113.

Gilmartin: I think you have to be careful, though, when you ask patients how they feel. You can't always rely on their subjective feelings because many patients will tell you that they're fine because you're their caregiver. I'm saying that to a physician or a nurse or a respiratory therapist, many patients will say, "Oh, I feel fine." But then you ask them five different ways and you find out that they have many symptoms. I have problems, always, with my patients in that respect because they say, "Well, I'm doing fine." But I look at them and I don't see that.

Bach: Campbell wasn't talking about medical symptoms, but we applied his anonymous surveying technique to severely disabled ventilator users and found that they considered themselves remarkably satisfied with life while healthcare professionals greatly underestimated their life satisfactions.[1,2]

REFERENCES

1. Bach JR, Campagnolo DI, Hoeman S. Life satisfaction of individuals with Duchenne muscular dystrophy using long-term mechanical ventilatory support. Am J Phys Med Rehabil 1991; 70(3):129-135.
2. Bach JR, Campagnolo DI. Psychosocial adjustment of post-poliomyelitis ventilator assisted individuals. Arch Phys Med Rehabil 1992;73(10):934-939.

Make: Or you may ask the patient's spouse or children.

Gilmartin: Yes, ask their spouse.

Make: Quality of life studies are based on patients' perceptions. But I think there is a potential flaw there in their methodology, even when using a standardized questionnaire. A spouse or other close relative may provide a different view of the patient. There's a questionnaire called the "Katz Adjustment Scale (KAS),"[1] which is given to patients' spouses or significant others to get their assessment. The KAS has a score that looks at the difference between the patient's and spouse's responses to see if there's concordance or discordance between their perceptions. So, it may be helpful to evaluate the perceptions of other people in the household.

REFERENCE

1. Katz MM, Lyerly SB. Methods of measuring adjustment and social behavior in the community: I. Rationale, descriptions, discriminative validity and scale development. Psychol Rep 1963;13:503-535.

Pierson: In this discussion, each of us is drawing on our own experience to feed into the subject matter and that experience is gained in our own practice setting. Barry practices at National Jewish, a tertiary or quaternary referral center; some of us practice in VA hospitals or in private clinics or in county hospitals. We may have to deal with patients who are homeless, patients who are substance abusers, patients who demand more healthcare resources than they need, "My headache is still there. I want an MRI." Yet, all of us sitting around this table are remarkably homogeneous in our cultural background. Just think about the context of this discussion, not only in different European countries, for example, but think about having this discussion in Japan or in India or in Nigeria. My point for making this comment is that this entire consideration of collaborative self-management has to occur within the cultural context in which we find ourselves. Unlike the strict biomedical model for healthcare, which we all agree is imperfect but which would be broadly applicable, this particular model for healthcare can only be considered within the narrower cultural context in which we happen to find ourselves.

Dunne: I always like to look for models that tend to eliminate the need to reinvent the wheel. I don't have much experience except for observations in how diabetes is managed with diabetologists and with nurse education. Does anybody at the table have an idea of how successful they are in managing Type I diabetes? I see an awful lot of success with this, in what is really collaborative self-management. Again, I don't have the empirical evidence, but it seems to me that might be a model in terms of how a patient is brought into managing a life-threatening situation, especially at the top end, with the Type I diabetes.

Make: I think the principles we've outlined are not unique to respiratory disease. If you look around the country, there's diabetes and hypertension (although, hypertension, I think, probably even more than diabetes is one in which there's a national effort to improve management of hypertension by having patients take a lot of responsibility because a lot of the management of hypertension is patient-controlled, it's not just taking medications, it's exercise and diet). So all of those behavior modification things are important. In fact, I haven't specifically reviewed patient education programs, but the information on patient education programs suggests that patient education is only helpful when the education is focused on what the patient has to do. I think that giving patients information about anatomy and physiology really is worthless because it doesn't achieve the desired outcomes. I think the problem is that you have to decide what the outcomes are that have to be achieved. The desired outcome may not be improving patient knowledge but improving patient behavior. So, patient education has to be behaviorally oriented to achieve some outcomes that we're looking for in order to be effective.

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Physiotherapy in Respiratory Care: A Problem-Solving Approach, by Alexandra Hough. Softcover, 243 pages, illustrated. London: Chapman & Hall, 1991. $29.95. Order through Singular Publishing Group 1-800-521-8545.

The author of this book, a Senior Lecturer for the North London School of Physiotherapy in England, states that effective treatment depends upon analytic problem solving and that this book was written for those who question fundamental assumptions and traditional rituals. The book is supposed to present both theory and practice and to emphasize a hands-on approach. It was written for audiences on both sides of the Atlantic and is intended for students or experienced physiotherapists and nurse specialists.

Physiotherapy in Respiratory Care contains 11 chapters, a glossary, 4 appendices, 13 pages of references, a suggested-reading list, and an index. Interspersed with the text are cartoons, illustrations, tables, photographs, radiographs, and interesting quotations from patients' experiences. References to published literature and to other sections of the book are cited throughout the text.

The first three chapters—"Overview of Physiology in Relation to Clinical Practice," "Assessment of the Pulmonary Patient" and "Respiratory Disorders"—cover 59 pages. In these chapters, a large number of topics are touched on, but not in much detail. The stated intent was to provide a "bedrock for problem-solving." I find the material interesting and informative, but do not believe that there is sufficient detail to adequately instruct an American respiratory-care student.

The next chapter, "General Management," covers, in 14 pages, prevention, oxygen therapy, nutrition, drug therapy, bronchoscopy, and rest. Once again, I find the material interesting and informative, but do not believe that there is sufficient detail to properly cover the material discussed. To be fair, English physiotherapists may only need to "know something about" these aspects of respiratory care. This may help to explain the lack of detail regarding respiratory care procedures in certain sections of this book. With respect to their need for respiratory care knowledge, it would seem that English physiotherapists are analogous to American physical therapists who provide chest physiotherapy.

In Chapters 5 and 6—"Overview of Physiotherapy Management" and "Physiotherapy for Specific Groups of People"—the author presents some of her strongest material. In Chapter 5 she covers methods to increase lung volume, to decrease the work of breathing, and to clear secretions; in Chapter 6 she covers topics such as dyspnea, rehabilitation, motivation, education, energy conservation, and physiotherapy in the home. I found many practical, hands-on examples of ways to provide chest physiotherapy—notes, tips, and suggestions for dealing with different types of patients.

In Chapters 8 and 9, "Intensive Care" and "Physiotherapy in Intensive Care," the author has again attempted to cover too much material. This of course may not be the case for a physical therapist who needs limited familiarity with mechanical ventilation, critical care monitoring, EKGs, and the immediate management of life-threatening cardiopulmonary disorders.

Chapter 10, "Physiotherapy for Neonates," covers, in only 9 pages, a comparison of neonates and adults, common respiratory disorders, general management, physiotherapy, and emergencies. This chapter also covers mechanical ventilation and CPAP.

Chapter 11, "Evaluation of Chest Physiotherapy," covers standards, evaluation of care, literature criticism, research, and continuing education. This is a good summary of what we should be doing to evaluate the care that we provide.

In general, I find the book easy to read and up to date. I did not have a problem with foreign phrases (of which there are a few), but a 4-page glossary of definitions, abbreviations, symbols, and values is provided, followed by a "Transatlantic dictionary" in Appendix A.

Throughout the book, the author does a good job of addressing how to deal with the needs, emotions, and rights of patients in a real-world hands-on fashion. In spite of my hesitation to recommend this book as a stand-alone text for respiratory care students, I believe that it does contain a lot of useful information and would be a valuable addition to any medical library.

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Learning Medical Terminology: A Worktext, 7th edition, by Miriam G Austrin BA RN and Harvey Austrin PhD. Hardcover, 543 pages, illustrated. St Louis: Mosby-Year Book Inc, 1991. $25.95.

Medical terminology can be intimidating to a student with no background in the medical sciences. The primary objective of this text is to enable the student to learn medical terminology, and I believe that this objective is accomplished. Once beginning students have completed the worktext, they should have a basic, workable vocabulary, applicable to any branch of medicine.

The seventh edition of **Learning Medical Terminology: A Worktext** is divided into four sections. In Section I, "The Basic Foundation of Medical Terminology," separate components of the words are broken down so that the student can understand the construction of medical words. This provides the necessary initial steps for building and formulating a medical vocabulary. For example, the essential components of our language (prefixes and suffixes) are arranged in chart form with meanings and examples. In addition, several helpful lists of prefixes, suffixes, roots, and combining forms are provided throughout the text, which cover actions, conditions, characteristics, body fluids and substances, chemicals, and colors.

Section II, "The Body Shell and Its Supports," introduces the medical language of the systems that support, move, and protect the body. This section begins with an excellent overview of cell composition, tissues, organs, and systems. Directional and anatomical planes are also discussed. I believe this provides the student with a good working knowledge with which to begin their study of the various systems. The skeletal, muscular and integumentary systems complete this section.

Section III, "The Internal Mechanism of the Body," consists of seven chapters that describe the internal workings of the body. Two of these chapters combine systems that are intimately related to each other (ie, the cardiovascular and lymphatic systems are paired as are the genitourinary system). A chapter on "the special senses" discusses the five senses and their unique structures and sensation mechanisms.

The last section, "Adding to the Structure," completes the building process by including enrichment material that cannot be isolated into individual systems; its only chapter is on the immune system and consists of a basic explanation of the immunologic process. The remainder of this section contains appendices of commonly used abbreviations and symbols, combining forms that relate to numbers, the metric system, English equivalents for linear measures, weights, volumes, and temperatures. Also, contained in this section is a list of medical specialty boards, such as the American Board of Internal Medicine.

To summarize, the external and internal anatomy are broken down by a systems approach, which ties basic explanations of each system of the body to relevant language. The authors use a short overview at the beginning of each chapter to present the material in an easily read format. A conviction that the immune system and its diseases will be of major concern in the 21st century is implied throughout this text. The glossaries of each chapter provide a phonetic pronunciation guide. Crossword puzzles and hidden word puzzles are also provided at the end of each chapter. These puzzles may be perceived as busywork by the advanced student, but may prove useful to the novice user of medical terminology.

Over 1,500 review questions and answers are included to help the student learn the content of the chapters. Sixteen full-color anatomic plates are located at the front of the book to further enhance learning. Instructors may find that the format allows programs to utilize the first four chapters alone as the basis for a short course in medical terminology, and to use additional chapters as an enrichment resource. An instructor's manual and audiotape pronunciation guides are available. Useful tips on teaching, including how to set up courses of different length, medical historical background, and a chapter test file with over 1,000 questions are also available.

There is a typo in the Preface of the book. According to the editor, the word "oncolory" should be "oncology." No other misspelled words were found.

In conclusion, **Learning Medical Terminology: A Worktext** is a well-conceived guide for the clinical student beginning in any healthcare specialty. The text is timely, well written, and attains its stated goal. I recommend this text as a guide to learning medical terminology.

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Exercise Testing and Exercise Prescription for Special Cases: Theoretical Basis and Clinical Application, 2nd edition, edited by James S Skinner MD, with 30 contributors. Soft-cover, 395 pages, illustrated. Philadelphia: Lea & Febiger, 1993. $37.50.

The study of exercise physiology and sports medicine, and its subsequent application to special groups of disease processes continues to grow and change with our ever-increasing level of knowledge. This second edition includes a complete revision for the chapters on cystic fibrosis and pregnancy. New chapters have been added on renal disease, asthma, low-back pain, and osteoporosis.

Thirty selected authors from several countries contributed to this text. Many of these authors are noted scientists and clinicians in exercise science who have also been involved in the evolution of the scientific information.

The book is divided into two major sections. The first section, composed of six chapters, discusses the general principles of exercise testing and prescription. It reviews the effects of gender, age, and environmental factors on exercise. We found this first section to serve as an excellent review for the clinician. Each chapter is well referenced, with additional reading suggested at the end of each chapter. Tables and graphs are presented throughout that complement the text and provide quick-reference guides to the reader.

In this section, the handling of oxygen consumption by gas analysis is confusing. Reference is made to the measurement of oxygen consumption by gas analysis in the first chapter, but then the authors go on to say it can be estimated from work load or power output measured during exercise. Later the authors refer to this direct measurement as the best method for assessing cardiac performance. Additionally, the authors state that measurement of oxygen consumption requires expensive equipment and technical support, but later refer to technical advances that make expired gas analysis easier to perform. The impression left with the reader may be one of confusion in terms of whether expired gas analysis has a place in the clinical environment. Perhaps the authors have a bias against using gas analysis measurements. Extensive literature supports the measurement of anaerobic threshold and maximum oxygen uptake in athletes and disease. Technology has advanced to a degree that makes this testing an integral part of many clinical programs. Perhaps the authors are more concerned about the need for standards and guidelines in this testing. The rest of the text in Section 1 refers to the use of anaerobic threshold and maximal oxygen uptake measurements in relation to both normals and specific disease states.

The second section is composed of 15 chapters that discuss exercise in specific disease states. Each chapter follows a similar layout, which includes the pathology of the disease, treatment, risk factors, how exercise affects the disease or the disease affects exercise, modification of exercise programs to meet the specific needs of the group, and the expected outcomes from the program. It is worthwhile to mention that in addition to the four new chapters and the updated chapters, other disease states covered include rheumatoid arthritis, diabetes, obesity, chronic obstructive respiratory disorders, coronary heart disease, hypertension, pediatric heart disease, valvular and congenital heart diseases in adults, and low functional capacity. Again, the authors of each chapter took great care in referencing the text and specifying when adequate scientific studies are lacking.

The sections on physiology and pathology in each disease process are general in nature. Their usefulness to the reader would be increased with a clearer focus on exercise application. Supportive evidence and clear indications for exercise therapy are lacking in some sections, particularly in diabetes, rheumatoid arthritis, and asthma. This may also reflect inadequate studies. The risks associated with exercise in specific disease states are well defined in some areas and unclear in others. We believe that clear supporting evidence and delineation of risk associated with each disease process must be provided if exercise therapy is to be provided safely.

The chapters devoted to exercise prescription in cardiac disease and chronic obstructive disease are excellent. This probably reflects the extensive research conducted on these topics and the authors' ability to present the information.

This text is an essential reference source for those in cardiopulmonary diagnostic laboratories, those responsible for exercise rehabilitation programs, and those physicians who prescribe and monitor exercise. It discusses the theory and application of exercise both in healthy subjects and those with disease. The number of references cited in each chapter (well over 100) makes this book an important addition to the exercise library. The authors have met their goal of providing a text with information for all professional groups who are interested in exercise and its interaction with our health status.

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English and Spanish: Medical Words and Phrases, by Minnie Rose, edited by Stanley Loeb, Executive Director, Editorial; Diane Labus and David Moreau, Editors; Maryann Foley RN BSN MEd, Clinical Consultant; Glafyra Ennis, Spanish Translation. Soft-cover, 177 pages. Springhouse PA: Springhouse Corp, 1994. $19.95.

As our national community becomes more of an *international* community, many healthcare agencies, facilities, and educational institutions —and the professionals who work in them—are finding it necessary to communicate with more non-English-speaking clients. Spanish is probably the most commonly encountered language other than English. **English and Spanish: Medical Words and Phrases** was compiled to assist healthcare workers in communicating with Spanish-speaking clients.

The book is formatted in an easy-to-read and easy-to-use manner. It starts with a section on the basics of Spanish pronunciation and grammar, which gives the novice Spanish-speaker a good start. The next section provides commonly used words and phrases that would be particularly useful in obtaining a medical history from the Spanish-speaking client.

Chapter 3 includes translations of common diagnostic tests, therapies, and treatments. Although many of the translations in this section will be understood by Spanish-speaking clients, the practitioner must remember not to use too much medical vernacular with laypersons. Regardless of the language that you are speaking, jargon is jargon.

The remaining chapters deal individually with the different body systems. Each chapter includes sections on current health problems, medical history, family history, health patterns, and psychosocial and developmental considerations. This chapter organization makes it easier for the reader to find a desired word or phrase.

The book also contains an appendix, which contains a list of drug classification translations. Again, the practitioner must beware of using excessive medical jargon with laypersons.

Generally, the book is a well-done, practical, easy-to-use reference. Although it does include some errors in translation that should be corrected, I believe that Spanish-speaking clients will still understand what is being said to them. While not an error, the authors list the formal translation for *you* as *usted*, which may be abbreviated as either *Ud* or *Vd*. The authors list only *Vd* as the abbreviation for *usted* then proceed to use *Ud* in the rest of the book. Readers would probably figure that one out for themselves, but it might cause some confusion initially. At any rate, it is a problem that should have been identified and corrected in the editing stages.

As previously mentioned, several errors in translation were noted. For example, on Page 18, the translation for *lie down* is given as *sientese Ud. Sientese Ud* actually means *sit down. Sientese Ud* is correctly given as the translation for *sit down* later in the chapter. The correct translation for *lie down—acuestese Ud*—is used in the translations for *lie on your back, lie on your side,* and *lie on your stomach.* On Page 63, the English word *how* is translated as *cuanto*, which actually means *how much.* The correct translation for *how* is *como.*

The book also contains several grammatical errors. The Spanish word *que* is misused in several areas, including Pages 54, 60, 65, 68, and 71. It is used in questions beginning with the English word *does. Que* is not necessary in these cases. Such sentences may be started with the main verb of the sentence. The authors use *que* correctly in other similar instances (eg, Pages 111 and 139). These editing errors detract from the overall quality of the book.

The concept behind this book is certainly a necessary one. Accurate communication with our clients, including those who speak Spanish, is crucial to quality care. With improved verification of translation and grammatical accuracy, **English and Spanish: Medical Words and Phrases** can be a useful aid for communicating with Spanish-speaking clients.

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CORRECTION

In the April issue of the Journal, an error was made on Page 313 of the paper by Patrick J Dunne "Demographics and Financial Impact of Home Respiratory Care" (Respir Care 1993(4);309-320). The sentence should have read "It is now conservatively estimated as many as 1,000,000 patients may be afflicted with ***severe*** COPD."

Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

AARC & AFFILIATES

May 31-June 3 in Myrtle Beach, South Carolina. The Georgia/South Carolina Region VI Committee presents its annual conference and assembly at the Landmark Hotel. Topics include Hospital Restructuring, Health Care Reform, Nitric Oxide, and Artificial Hemoglobin. Contact Lynda Thomas-Goodfellow at (404) 651-1498, or write to Region VI Committee, 275L Harbison Blvd, Suite #2, Columbia SC 29212.

May 31-June 3 in Orlando, Florida. The FSRC presents the annual Sunshine State Seminar in Orlando, Florida. This 4-day seminar provides topics from critical care to patient-focused care, with special exploration of topics related to HIV/AIDS. CEUs have been applied for, and a 4-day participant can receive 21 CEUs. Our social calendar includes, among other things, a golf tournament, and an evening at Sea World. Contact Marlene Thomas (904) 734-3320, ext 1290, or fax (904) 738-3102.

June 1–3 Steamboat Springs, Colorado. The CSRC presents its annual convention at the Sheridan Inn. This year's theme is "Respiratory Care Everywhere in Health Care" and the opening keynote speaker is Sam Giordano, MBA, RRT, Executive Director of the AARC. Giordano speaks on "The Effect of Health Care Reform on the RCP." Topics include pulmonary rehabilitation, clinical pathways, gas exchange, noninvasive mechanical ventilation, pulmonary laser therapy for emphysema, and permissive hypercapnia. Other activities include the annual golf tournament, a picnic, the Sputum Bowl finals, and an awards presentation. Contact Shelly Collings at (303) 770-2220.

June 1-3 in Des Moines, Iowa. The ISRC annual conference is held at Hotel Savery in downtown Des Moines. Keynote speaker Senator Charles Grassley addresses health care reform. Contact Mike Wheeler (515) 263-5108.

June 8–10 in Round Top, New York. The Seventh Annual Managers /Educators Rocking Chair Conference, at the Winter Clove Inn and Conference Center, is sponsored by the NJSRC, NYSSRC, AARC, and Allied Health Care Services of Orange, NJ. The conference, offered to all respiratory managers, supervisors, and educators, features Sherry Milligan, Associate Executive Director of the AARC, and Carl Wiezalis of SUNY Health Science Center. Topics include national health care reform and its impact on healthcare delivery, marketing the RCP, preparing for on-site inspections from JRCRTE and JCAHO, and protocols. The registration fee is $225, which includes conference registration, lodging for two nights, meals, and gratuities. The same package with one night's lodging is $175. Contact Ken Wyka at (201) 982-7228 or Joseph Sorbello at (315) 464-6872.

June 8-10 in Checotah, Oklahoma. The OSRC presents its 29th Annual State Seminar and Exhibition at Fountainhead Resort and Convention Center on Lake Eufaula. This year's theme, "The Times–They Are A' Changing," addresses anticipated changes in the healthcare delivery system and critical issues for respiratory care. Other activities include a Manager's Mixer and the traditional golf tournament. For information on the seminar or exhibits, contact Karen Hart RRT at (405) 273-6442

June 15-17 in St Charles, Illinois. The ISRC presents its 26th annual state convention at Pheasant Run Resort. This year's theme is "ISRC Game Plan: Team Spirit '94" and features Sam Giordano MBA RRT, Executive Director of the AARC, as the keynote speaker. Mr Giordano speaks on "Health Care Reform and the RCP." New this year are breakfast sessions with choices of four topics: "CLIA Update," "OSHA Update," "Hospital-Based Nicotine Intervention Program," and "Reimbursement Issues for Home Medical Equipment Suppliers." The convention includes many other educational topics, an entry level review, and a resort setting for your enjoyment and relaxation. Contact Douglas McQueary (312) 962-4060 or Jane Reynolds (312) 883-6535.

OTHER MEETINGS

June 10–12 in Memphis, Tennessee. St Joseph Hospital and Health Centers presents its 14th Annual Respiratory Care Symposium, a program designed to prepare candidates for the CRTT exam. Contact Konnye Warren or Dot Younginer, St Joseph Hospital, 220 Overton, Memphis TN 38105. (901) 577-2780.

August 31-September 3 in Christchurch, New Zealand. The Third Congress of the Asia Pacific Association for Respiratory Care presents "Breathing into 2001," at the Christchurch Town Hall, Christchurch, New Zealand. Contact Stephanie K Humphries, The Planit Group Limited, Event Organizers, 201 Cambridge Terrace, Christchurch, New Zealand. (643) 366-5955, fax (643) 366-5944.

Final Call
Deadline May 28

1994 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Las Vegas, Nevada, December 10-13, 1994. Accepted abstracts will be published in the November 1994 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) an **original study,** (2) the **evaluation of a method or device,** or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. A **method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience *or* a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. A **case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include: (1) patient data case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form *and simple figures may be included provided they fit within the space allotted. No* figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested in right column of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts received by March 15 will be reviewed and the authors notified by April 22. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (May 28).

Final Deadline

The mandatory Final Deadline is May 28 (postmark). Authors will be notified of acceptance or rejection by *letter only*— to be mailed by August 15.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

1994 RESPIRATORY CARE OPEN FORUM

Abstract Form

22.2 cm or 8.8"

8.1 cm or 3.2"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with *all* authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave 'ragged' right margin).
4. Do not use type size less than 9 points.
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

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Presenter's Mailing Address

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11030 Ables Lane
Dallas TX 75229

Early deadline is March 15, 1994
(abstract received)
Final deadline is May 28, 1994
(abstract postmarked)

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Notices

The American Respiratory Care Foundation Awards for 1994

Allen & Hanbury Literary Awards

$2,000 for the best original paper (study, evaluation, or case report) accepted for publication from December 1993 through October 1994. This award is not limited to papers based on OPEN FORUM presentations.

Four awards of $1,000 each for papers accepted for publication from November 1993 through October 1994 based on any OPEN FORUM presentation (not limited to 1993 OPEN FORUM).

Five awards of $500 each for the best papers submitted (not necessarily published) by 1994 OPEN FORUM participants who have 'never published' in the Journal. The never-published first author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the 1994 Annual Meeting (received in the Editorial Office by November 1, 1994). Co-authors may have previously published in RESPIRATORY CARE.

Dr Allen DeVilbiss Literary Award

$2,000 cash plus travel expenses to the AARC Annual Meeting to receive the award—for the best paper published from November 1993 through October 1994 that addresses new technology or a new application of current technology in respiratory care.

Radiometer America Literary Awards

Three awards of $333 each are to be awarded to the authors of the three best features from Test Your Radiologic Skill, Blood Gas Corner, Kittredge's Corner, and PFT Corner accepted for publication from November 1993 through October 1994. All three (or none) of the features may be chosen from a specific category (eg, all three may be chosen from Blood Gas Corner).

OPEN FORUM 1994

It's last call to submit your abstracts for possible presentation at the OPEN FORUM during the AARC Annual Meeting in Las Vegas, Nevada, December 10-13, 1994. The final deadline is May 28; abstracts must be postmarked by May 28 to be accepted for review. For more information on changes to the rules/instructions, see Page 405 of this issue.

Annual Meeting Registration Reimbursement

As in the past, any 1994 OPEN FORUM presenter (or co-author designee) will receive complimentary registration for an adequately prepared paper based on his 1994 OPEN FORUM abstract, submitted prior to or at the 1994 Annual Meeting. (If submitted on-site and accepted for review, convention registration will be reimbursed.)

All awards will be made at the 1994 Annual Meeting. Papers are judged automatically. No application is necessary.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1994 Examination and Fee Schedule

	Examination Date	Application Deadline
CRTT:	July 16	May 1
	November 12	September 1
RRT:	June 4	February 1
	December 3	August 1
CPFT:	June 4	April 1
RPFT:	December 3	September 1

Fee Schedule

Entry Level CRTT—new applicant:	$190.00
Entry Level CRTT—reapplicant:	$160.00
RRT Written and Clinical Simulation—new applicant:	$190.00
Written Registry Only—new applicant:	$ 90.00
Written Registry Only—reapplicant:	$ 60.00
Clinical Simulation Only—new & reapplicant	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00

	Active	Inactive
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RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P Specialty Credentialing:	$25.00	$130.00
Membership Renewal: CRTT/RRT/CPFT/RPFT		$ 12.00

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B ☐ Cardiopulmonary
C. ☐ Anesthesia Service
D ☐ Emergency Dept
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2. ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5. ☐ Pulmonary Function Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management
IV. Position
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B ☐ Chief Therapist
C. ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
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G ☐ Medical Director
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I ☐ Pulmonologist
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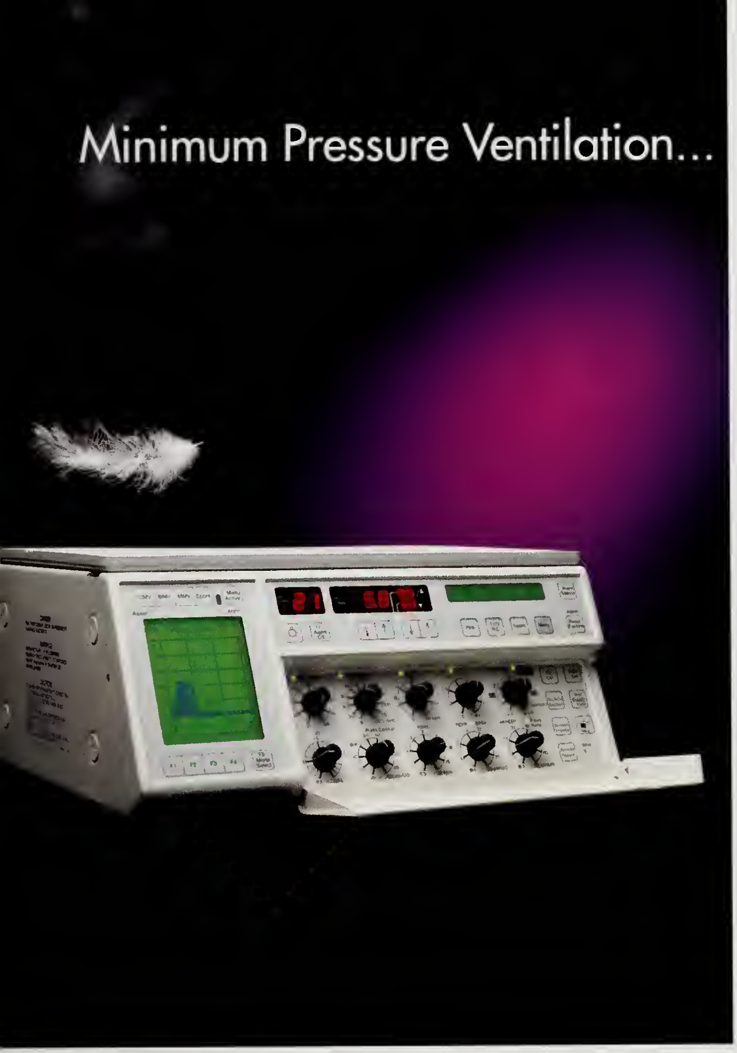

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